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Studies in Periodontal Pathology

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A thesis submitted in conformity with the requirements for the Degree of Doctor of Philosophy in the University of Toronto.

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INTRODUCTION

NO adequate understanding of periodontal pathology can be attained except through a comprehension of the correlation which exists between structure and function. In arriving at a broad conception of the subject as a whole, the functions of the teeth and their supporting structures form the basis for the interpretation of the information gained through research in the field of the normal and pathologic anatomy of these regions. In other words, the study of physiology must go hand in hand with the study of histology and histo-pathology. Histo-pathologic research, as is to be expected, has a most direct and intimate bearing upon the scientific and successful investigation of periodontal disease. And a careful consideration of the findings of the microscope is essential in the formulation of correct theories.

The importance of histo-pathology in the field of periodontia need not be emphasized, as it is a self-evidenced fact that scientific diagnosis must be based upon an appreciation of this science. There is, however, on the part of many scientists, a growing realization that the evidence of the clinic furnishes a contribution to the whole body of knowledge which must also be given consideration. And this holds for periodontology as well as for other fields in science.

In this paper we shall concern ourselves chiefly with the morphologic changes which take place during the development of the lesions commonly found in the periodontal tissues. In presenting the histopathology of the periapical together with the gingival and parietal tissues, emphasis is laid on the fact that periodontology must of necessity embrace the consideration of all the tissues which lie in proximity to the tooth. Furthermore, disease inaugurated in one part of the periodontium may, and frequently does, extend to and involve every part of this complex structure. Hence one part of the periodontium must not be viewed to the exclusion of the remainder.

Note: A limited number of Bulletins, covering Dr. Box's previous work on the Dentinal-Cemental Junction and Normal and Pathological Histology of the Dental Pulp, will be supplied upon application.—Canadian Dental Research Foundation.

PART I: NORMAL HISTOLOGY.

A fundamental knowledge of periodontal disease is impossible without an insight into the normal histological characteristics of the tissues in which the pathological changes are initiated and carried on. The following brief description of the minute anatomy of the periodontium, e.g., the gingivae, the pericementum, and the alveolar process, will therefore be in order.

THE GINGIVAE.

The gingivae include all the soft tissues which invest the cervical portions of the crowns and roots of the teeth, to the level of the crests of the alveolar process. The main bulk of the gingival tissues rests upon the crests of the alveolar process and is supported by them. Attached to the cementum, from the level of the alveolar crest to the gingival line, which is marked by the amelo-cemental junction, is the division of these tissues, known as the cemental gingivae. The cemental gingivae are attached to the alveolar crest by fibres of the periosteum and certain principal fibres which are inserted in the cementum.

Rising from the cemental gingivae and supported by them, are wedged-shaped tissue extensions which encircle the enamel at the cervix. These are known as the marginal gingivae. By their contact with the enamel, they form the so-called gingival crevice, the deepest portion of which is bounded by the first attachment of the cemental gingivae at the amelo-cemental junction (fig. 2).

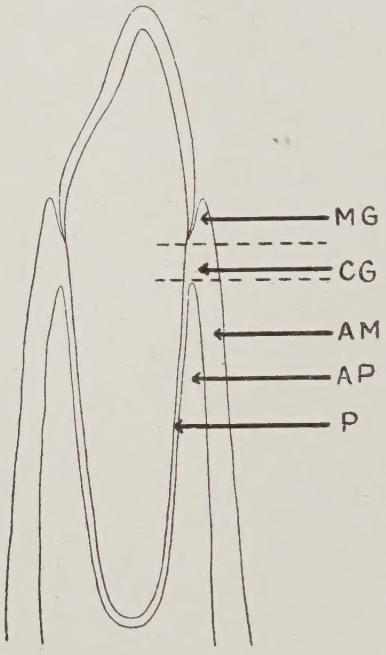

Fig. 1.—Diagrammatic drawing showing the divisions of the periodontal structures.

M G=Marginal gingiva. C G=Cemental gingiva. A M=Alveolar mucosa. A P=Alveolar process. P=Pericementum.

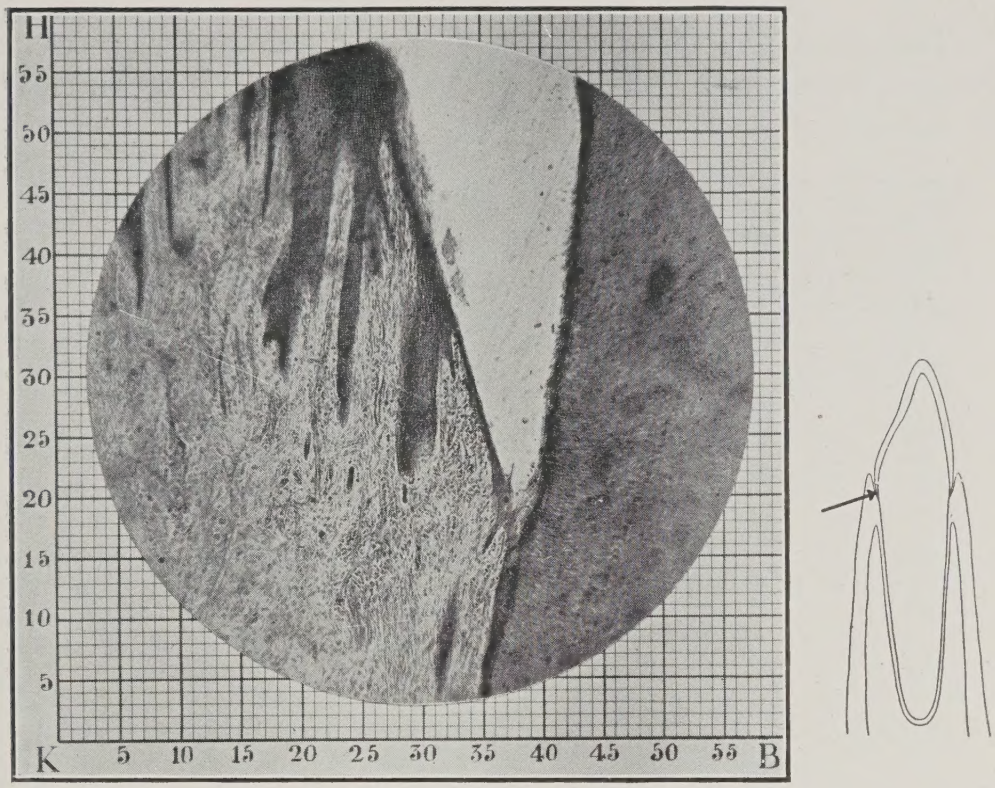

Fig. 2— x 94. Marginal and cemental gingivae, showing the base of the gingival crevice. Note the extremely thin layer of epithelium on the crevicular surface of the marginal gingiva in this specimen. The epithelium is that portion of the marginal gingiva shown in the illustration as very dark in comparison with the connective tissue.

H K 18 x K B 38 (see explanatory note on opposite page), Base of gingival crevice. Tissue above line H K 18 is the marginal gingiva; tissue below this line is the cemental gingiva.

H K 30 x K B 34, Extremely thin layer of crevicular epithelium.

H K 40 x K B 23, Connective tissue papilla.

An explanation of the method of mounting the photographs in this paper may be in order here.

It has long been the practice in plane geometry to locate the position of any point, line or surface, by reference to a system of two fixed lines, usually at right angles to each other, called the axes of coordinates. This is exactly similar to the method of indicating a point on the earth's surface by a statement of its latitude and longitude. These coordinates intersect at a point called the origin, and distances above or below the horizontal or base line are usually denominated ordinates, while distances to one side or the other of the vertical line are termed abscissae.

The common practice of indicating any special point on a photomicrograph by drawing a circle round it or placing a letter close to it is objectionable because it mars the photograph as a whole and quite often obscures something that a reader may wish to see, also it is not always precise. The method of providing a separate skeleton drawing of the photograph and placing the same over or beside the original complicates the reference. For these reasons it was decided to mount the photographs in this article on a coordinate mat so that any point in the photograph could be located by stating its coordinates.

Two lines intersecting at the point K and denominated HK and KB form the axes of reference and lines parallel to these are drawn to the number of 60. This forms the mat upon which all the photographs are mounted. It then becomes an easy matter for the reader to locate any

The name alveolar mucosa is given to the tissue which covers the alveolar process for a variable distance apically. It is continuous with the cemental gingiva.

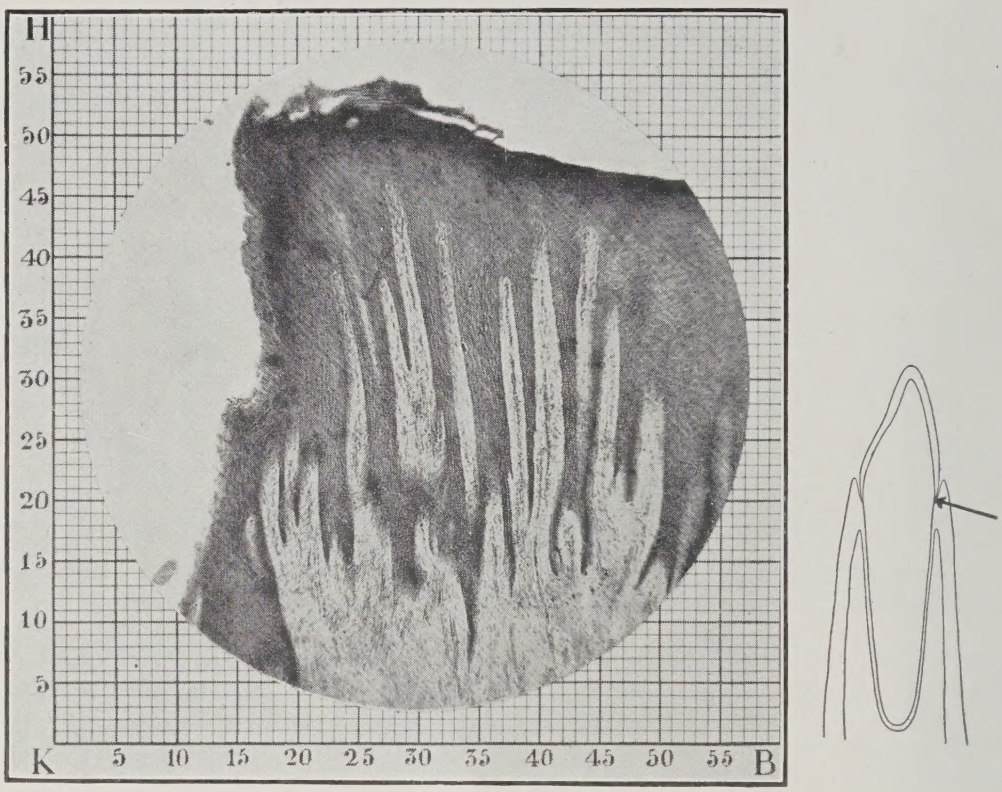

Fig. 3— x 94. Marginal gingiva, showing thick epithelial covering on the crest and external surface: also deep invagination of the epithelium by connective-tissue papillae.

H K 50 x K B 20, Crest of marginal gingiva. Epithelium extends from level of H K 15 to H K 52.

H K 30 x K B 38, Typical connective-tissue papilla.

H K 35 x K B 10, Gingival crevice.

The epithelium which covers the gingivae is of the stratified squamous type, and rests on a thin basement membrane. It differs from that of the alveolar mucosa in that it becomes thicker and stronger progressively from a point approximately opposite the crest of the alveolar process, to the crest of the marginal gingiva. A characteristic of the gingival epithelium is its invagination by finger-like

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point referred to in the text by the simple expedient of laying a straight edge across the photograph at the distances indicated.

Professor G. R. Anderson, Professor of Engineering Physics, University of Toronto, and Professor of Physics, Royal College of Dental Surgeons, suggested the possibility of utilizing this system of co-ordinates in the minute description of photomicrographs. He planned and drafted the system used in the accompanying illustrations. Prof. Anderson also made the photomicrographs for each of the papers published in this issue. To this work he brought an intimate and far-reaching knowledge of the physics of light and of photography, and by the perfection of the results thus obtained, has placed the author under a lasting obligation which is hereby gratefully acknowledged.

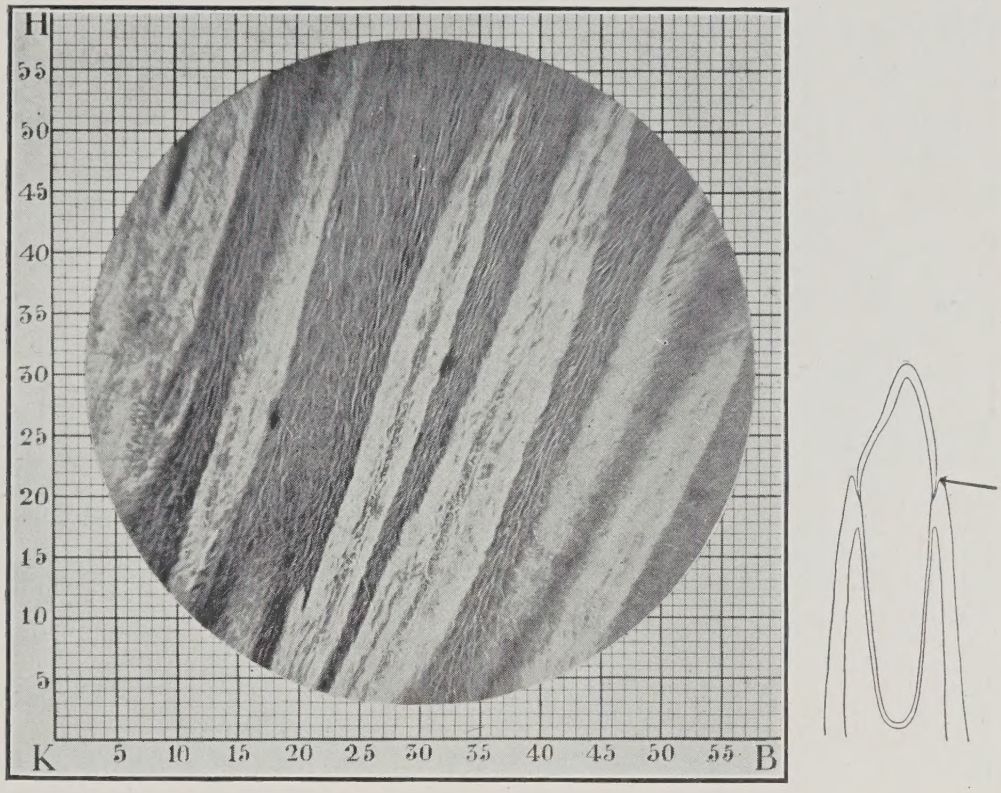


Fig. 4— x 250. Connective-tissue papillae in the epithelium of the marginal and cemental gingivae showing blood-vessels.

H K 53 x K B 38, Tip of one of the papillae.
H K 38 x K B 40, Another connective-tissue papilla.
H K 23 x K B 34, Blood-vessel in papilla.

papillae of connective tissue (fig. 4). The distribution of the epithelium on the crevicular surface of the marginal gingiva is worthy of special note because, as a rule, it is unusually thin. Occasionally breaks can be noticed in this lining, where the epithelium is absent, and again, imperfect junctions of the crevicular epithelium with the cementum are frequently observed. These facts,—its extreme thinness, the possible presence of breaks on the surface and imperfect junctions—render this crevicular surface the weakest link in the chain of the epithelial cordon in the mouth.

The gingivae have an extremely rich blood supply, which is derived from the pericemental and periosteal blood-vessels, as well as from those of the adjoining alveolar mucosa. The connective tissue of each papilla carries an arteriole which passes to its extremity, where it terminates in a capillary loop and returns as a vein (figs. 5 and 6). This circulatory apparatus is designed to carry nourishment to all the epithelial cells. It is frequently seen, in the microscopic study of these tissues, that the cells separating the capillary loops from the external surface, and hence from the mouth flora, are very few in number (figs. 7, 8, 9, 10, 11, 12).

The alveolar mucosa is characterized by a thin epithelium with shallow, invagination by connective tissue papillae, and by a connec-

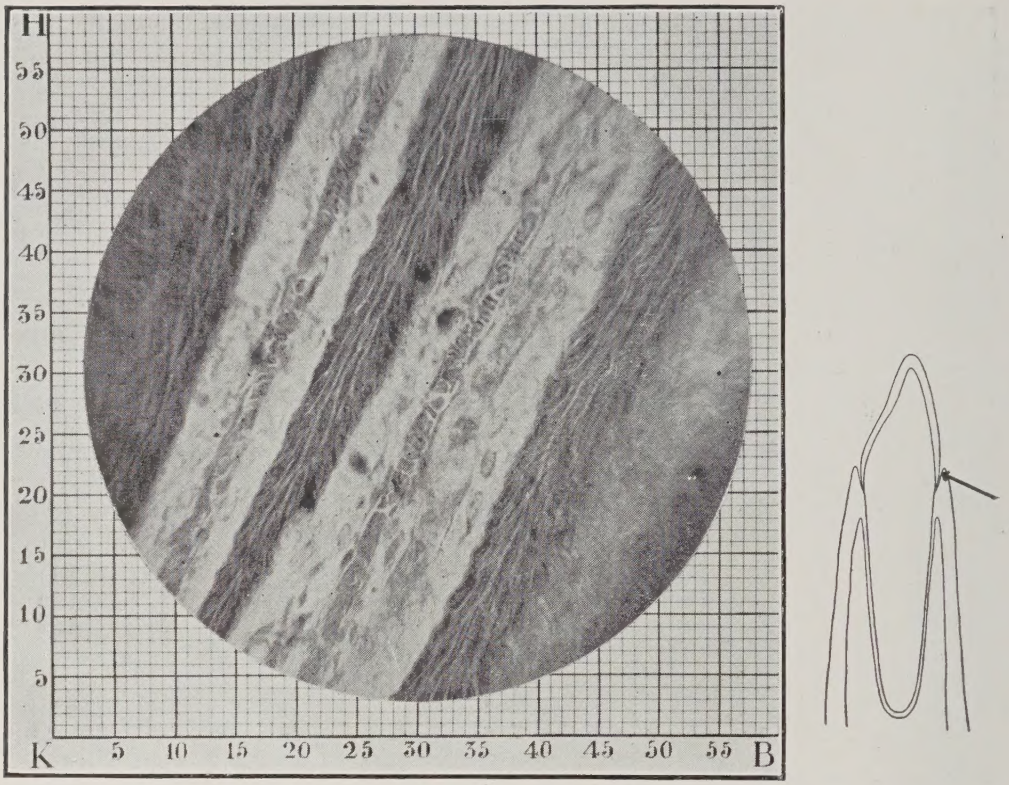


Fig. 5— x 500. Two connective-tissue papillae, showing arteriols and venule. The smaller papilla has a width of 9 (coordinate) lines, the larger has a width of 14 (coordinate) lines.

H K 33 x K B 34, Arteriole. Note the red blood-cells in the arteriole.
H K 33 x K B 37, Venule.

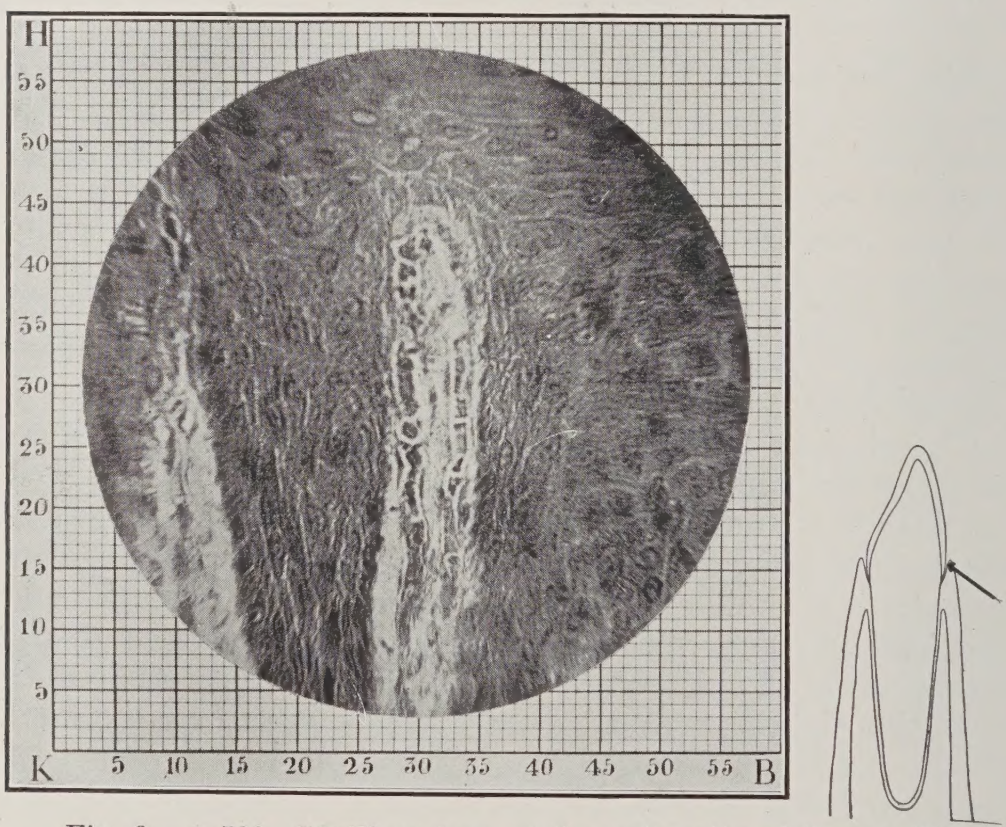


Fig. 6— x 500. Capillary loop at the extremity of the papilla.

H K 44 x K B 31, Capillary loop.
H K 50 x K B 31 Stratified squamous epithelium.

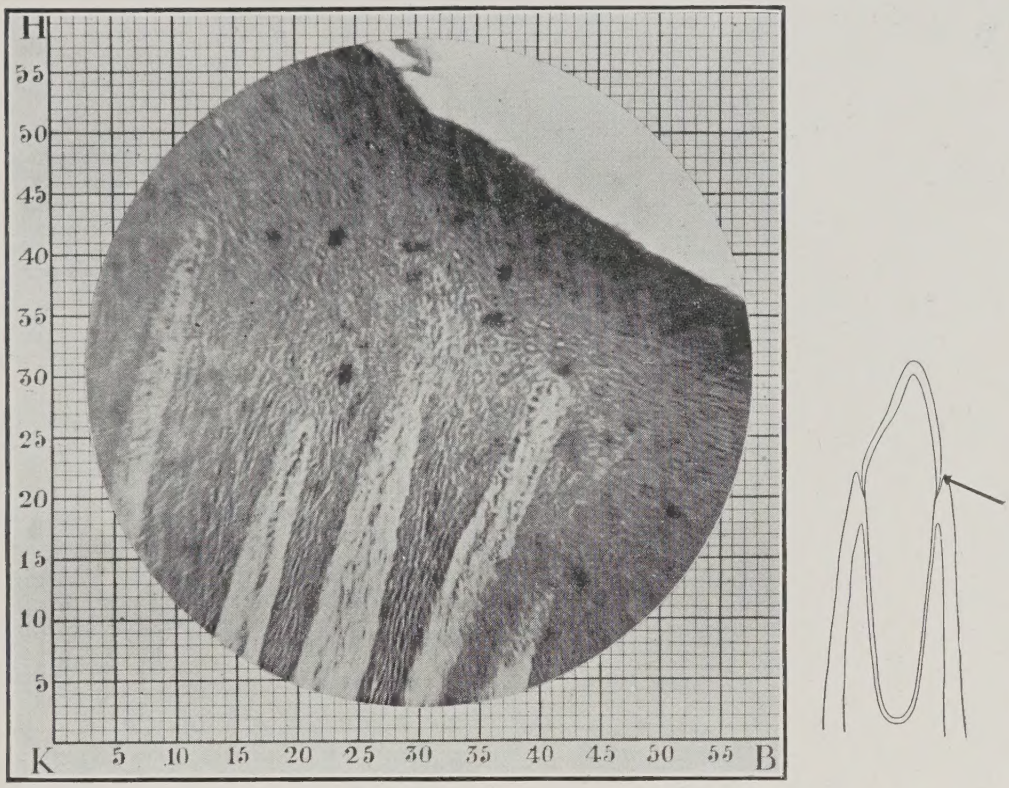


Fig. 7— x 250. External surface of the marginal gingiva, showing proximity of the capillary loops in the papillae, to the surface. The layer of epithelial cells protecting the blood-stream from mouth bacteria is here quite thin.

H K 31 x K B 41, Extremity of papilla, containing capillary loop.
H K 42 x K B 46, External surface of marginal gingiva nearest this point.

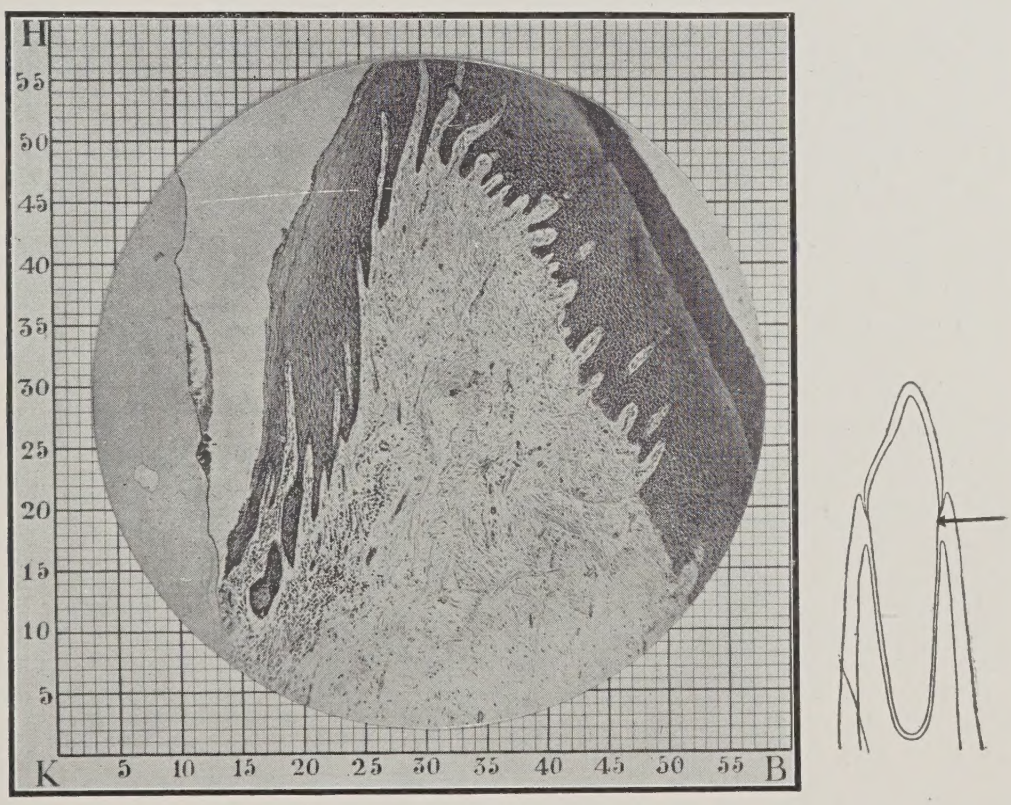
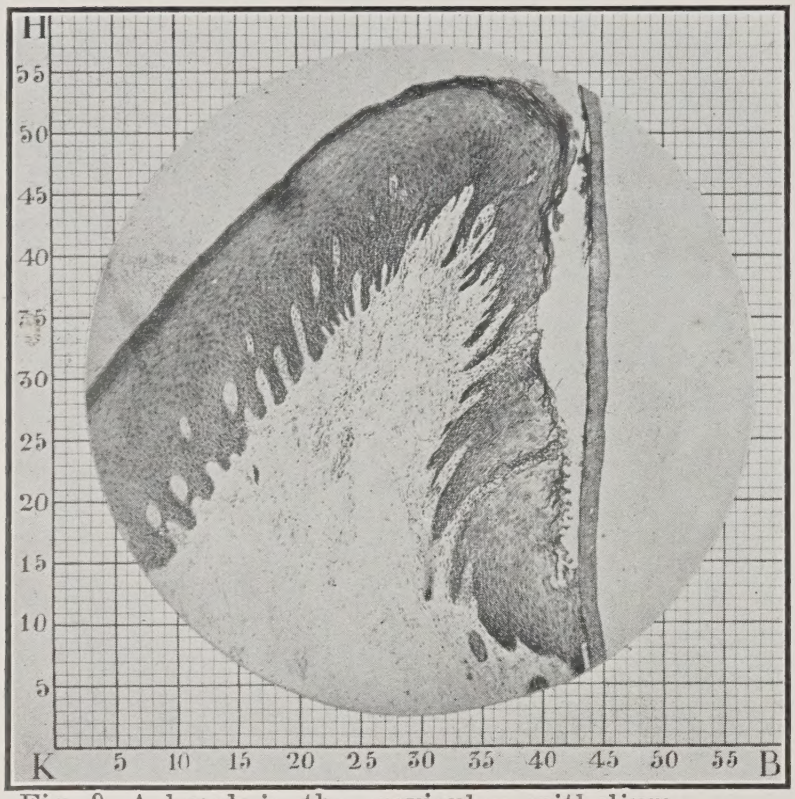


Fig. 8. Faulty junction of the crevicular epithelium with the tooth.

H K 15 x K B 13, Base of gingival crevice. Note lack of epithelial protection of connective tissue of cemental gingiva from the contents of the gingival crevice. Note also small area of leukocytic infiltration in adjacent connective tissue. H K 40 x K B 13, Gingival crevice.



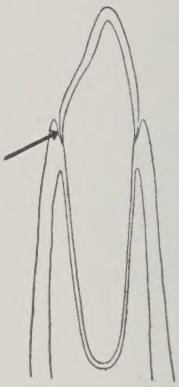
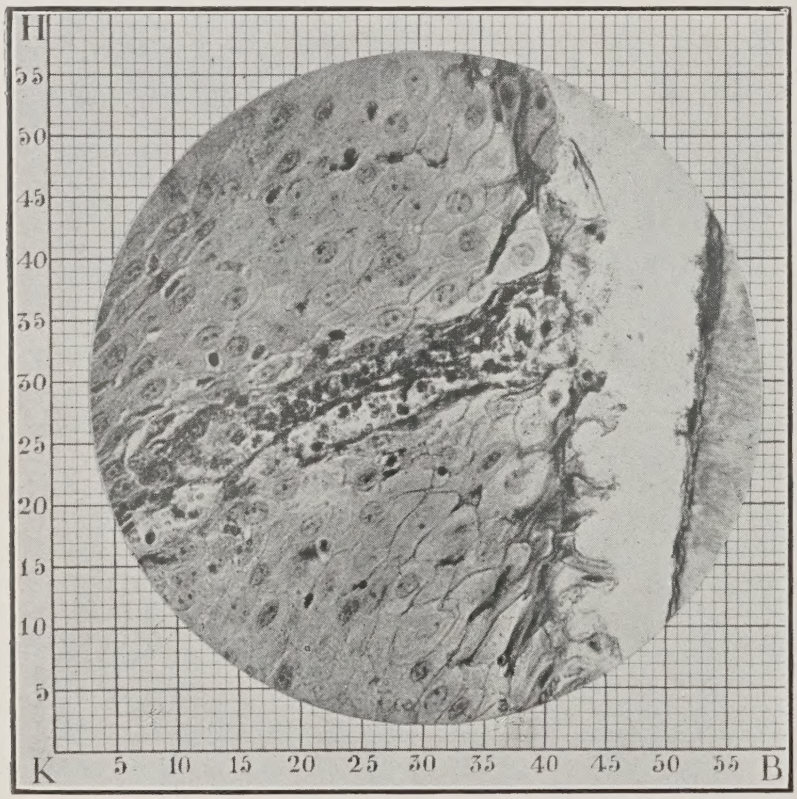


Fig. 9. A break in the crevicular epithelium.

H K 26 x K B 41, Connective tissue of marginal gingiva exposed to the fluids of the gingival crevice. H K 36 x K B 42, Gingival crevice.



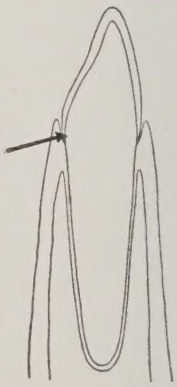


Fig. 10— x 500. High magnification of the break in the crevicular epithelium shown in the previous illustration.

H K 36 x K B 43, Break in epithelium.

H K 30 x K B 23, Blood-vessel in connective tissue. Note that the blood-vessel extends nearly to the crevicular surface.

H K 35 x K B 47, Gingival crevice.

H K 40 x K B 54, Bacteria on surface of tooth.

H K 45 x K B 33, Nucleus of epithelial cell. Note well defined outlines of epithelial cells and of nuclei.

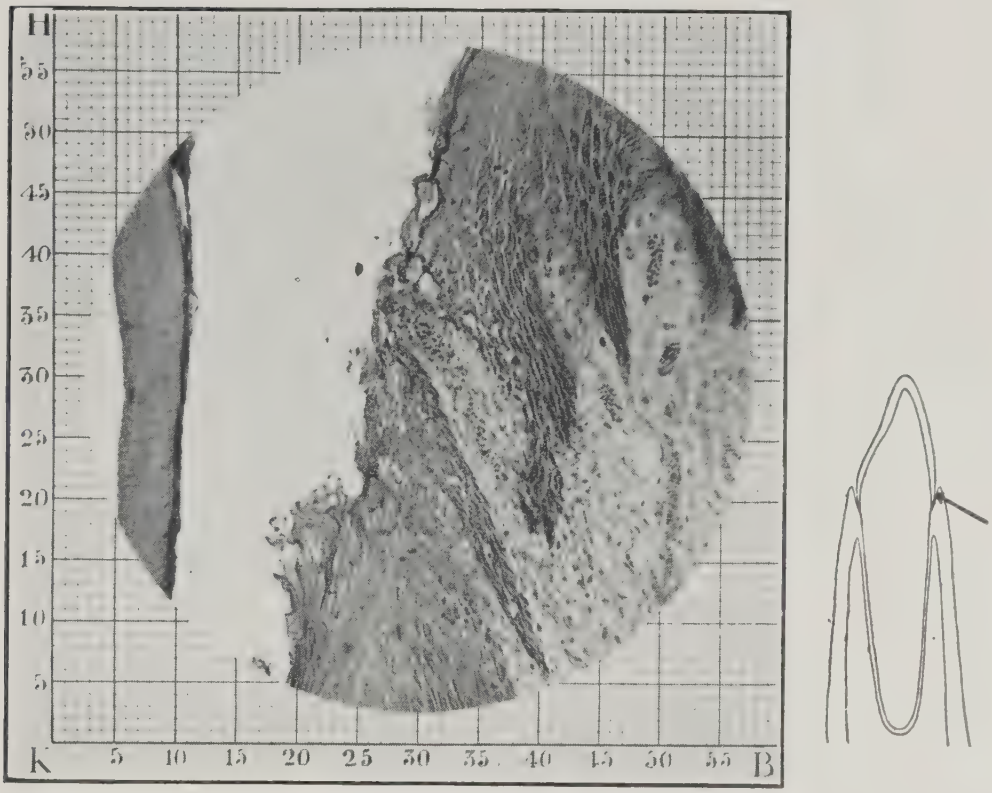


Fig. 11. A break in the epithelium on the crevicular surface. Note the blood-vessel passing to the surface.

H K 37 x K B 27, Break in epithelium.

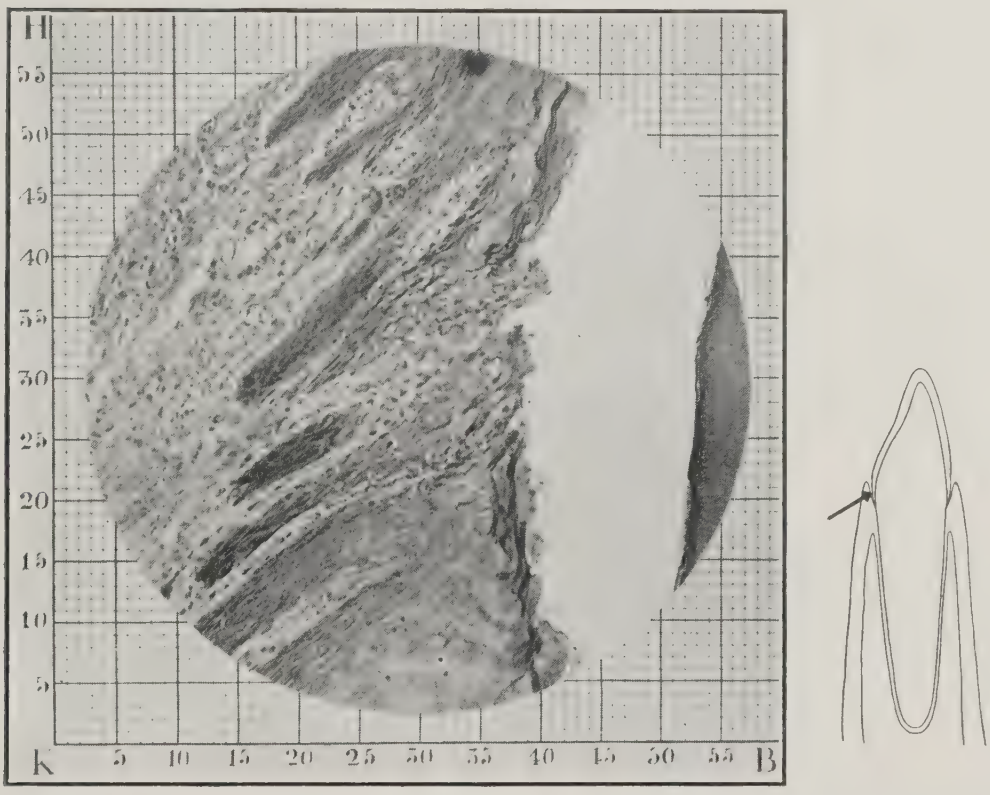


Fig. 12. Group of breaks in the epithelium on the crevicular surface.

H K 42 x K B 40 to H K 25 x K B 39, Total extent of break.

tive tissue which shows a dense interlacing meshwork of fibres of white connective tissue. This latter feature gives to this tissue its comparative immobility (fig. 13).

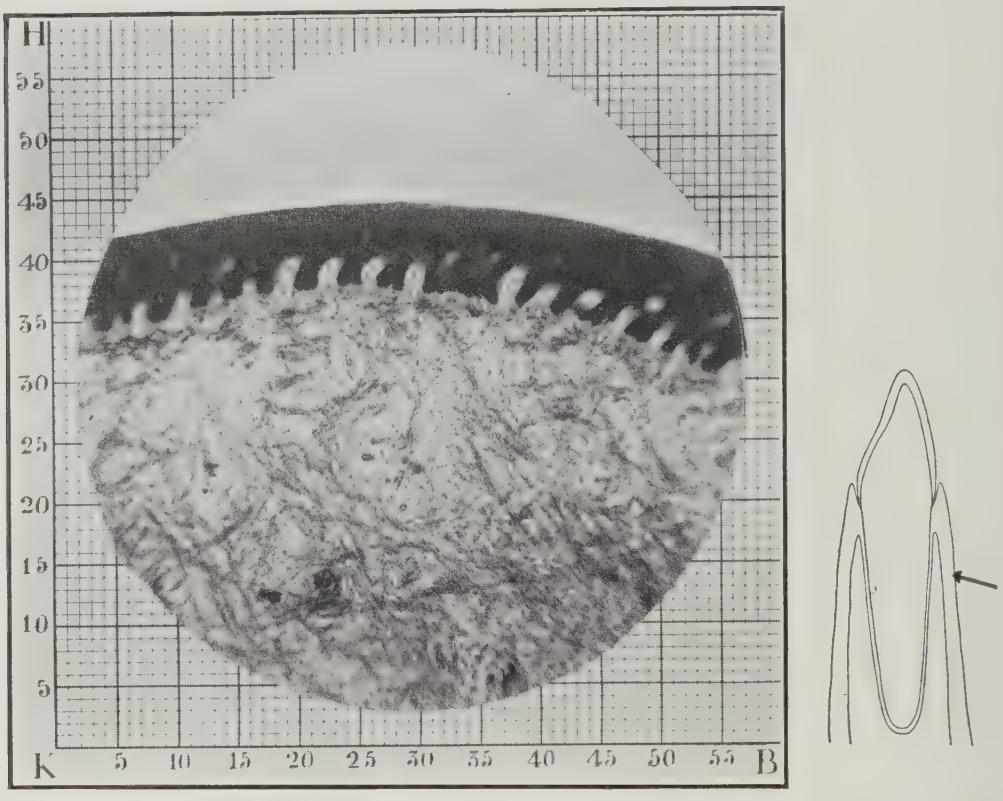


Fig. 13— x 94. Alveolar mucosa, showing comparatively thin epithelium, also the interlacing connective-tissue fibres which bind it to the periosteum.

H K 43 x K B 30, Stratified squamous epithelium. The connective-tissue fibres are shown in the lower part of the illustration.

Having their insertion in the cementum and extending into the marginal gingiva, certain fibres of the white connective tissue variety forming a fairly definite grouping, can be demonstrated. They comprise what is known as the marginal group (fig. 14). Passing mesiodistally from tooth to tooth, through the cemental gingivae, is found another definite grouping of fibres known as the trans-septal group (fig. 15).

PERICEMENTUM.

The pericementum is the tissue that fills the space between the root of the tooth and the wall of the alveolus. It attaches the teeth to the alveolar process. In the study of the histological structures which constitute this important tissue this order will be followed:

1. Principal fibres.
2. Fibroblasts.
3. Cementoblasts.
4. Osteoblasts.

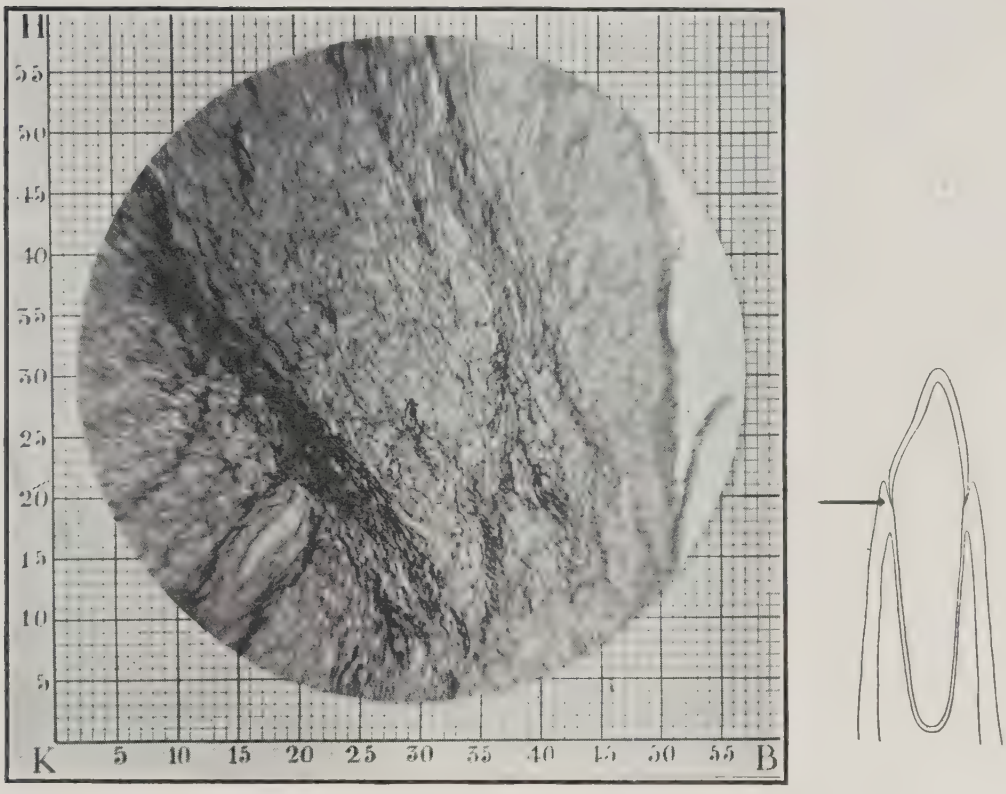


Fig. 14— x 187. Marginal group of principal fibres in the marginal gingiva.

H K 20 x K B 51, Base of gingival crevice.
H K 35 x K B 50, Crevicular epithelium.
H K 23 x K B 24 } Two bundles of principal fibres belonging to the
H K 38 x K B 39 } marginal group.

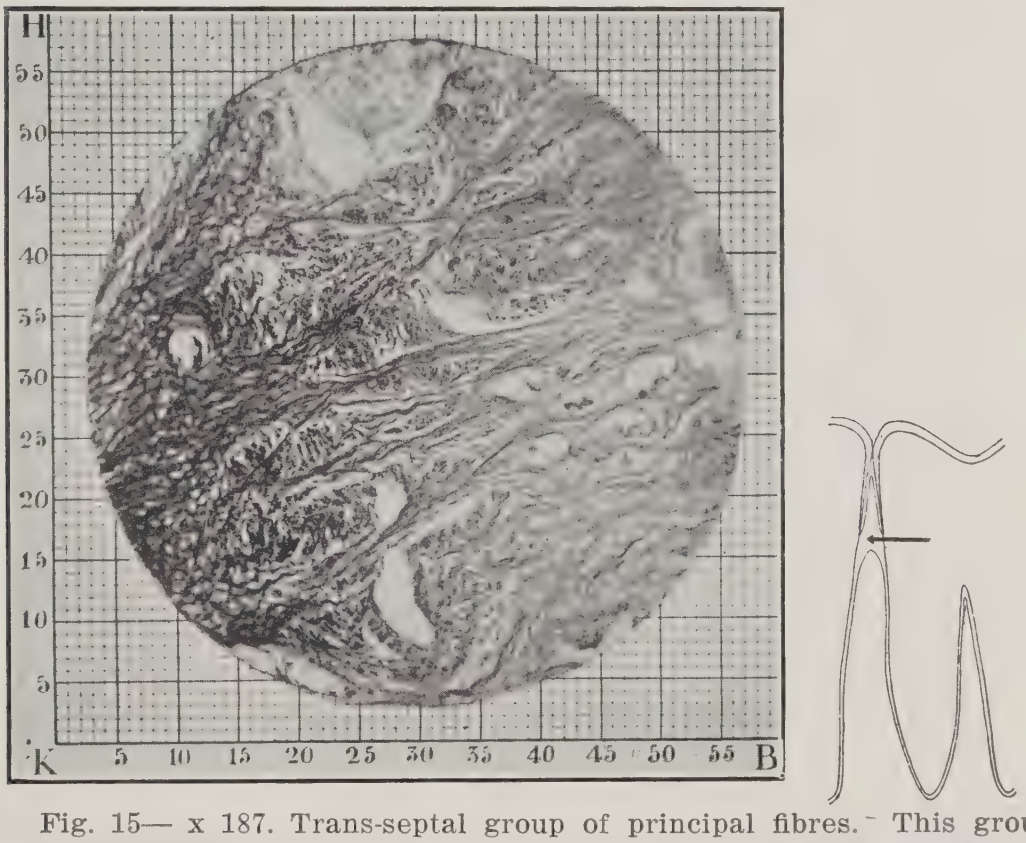


Fig. 15— x 187. Trans-septal group of principal fibres. This group passes mesiodistally from tooth to tooth through the cemental gingiva.

H K 30 x K B 18 } Bundles of principal fibres in the cemental gingiva.
H K 35 x K B 45 }

5. Osteoclasts.
6. Epithelial structures.
7. Blood-vessels.
8. Nerves.

1. *Principal Fibres.*

These fibres which compose the greater part of the pericementum are of the white connective-tissue variety and are attached to the cementum on one side and on the other extremity, depending on the location, to the connective tissues in the marginal gingiva, the fibrous mat of the alveolar mucosa, the periosteum of the alveolar process, the cementum of the approximating tooth or the bone of the alveolar process. While these fibres, as a whole, maintain a uniform progression from the crest of the process to the cemental apex, yet they exhibit a fairly definite tendency to become associated into groups. These fibre-groupings have been well described by Black. Beginning at the crest of the alveolar process and progressing apically, these groupings are as follows:

1. The alveolar crest group consisting of those fibres which pass over to, and are inserted into the crest of the alveolar process (figs. 16 and 17).

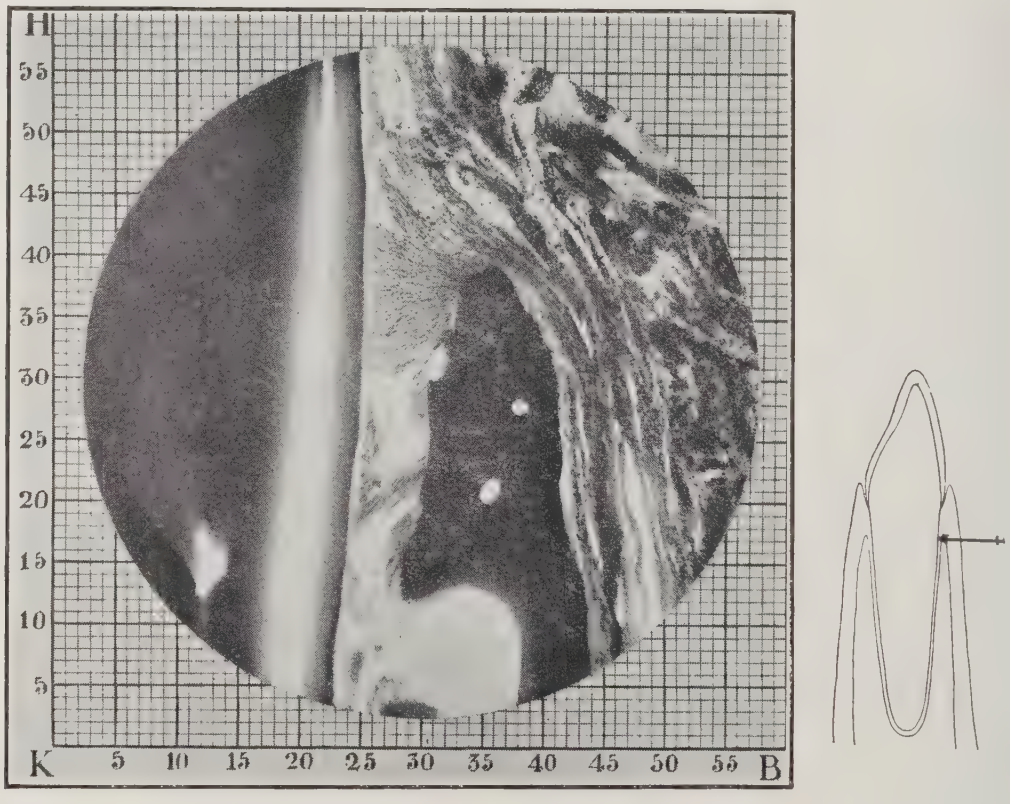


Fig. 16. Alveolar crest group of principal fibres. Note the fan-shaped arrangement of the fibres as they pass from the alveolar crest to their insertion in the cementum.

H K 38 x K B 36, Crest of alveolar process.
H K 39 x K B 29, Alveolar crest group of principal fibres.
H K 51 x K B 25 to H K 30 x K B 25, Insertion of principal fibres of this group into cementum.
H K 21 x K B 27, Pericementum.

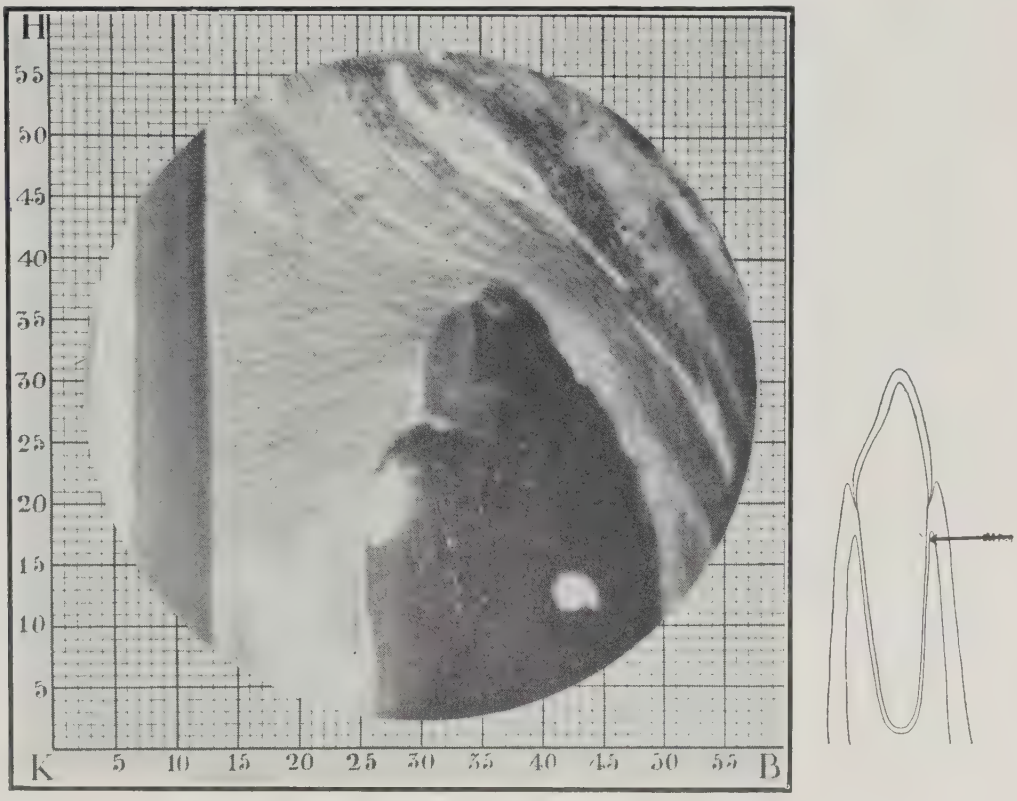


Fig. 17— x 187. Higher magnification of the alveolar crest group of principal fibres.

H K 37 x K B 37, Crest of alveolar process.
H K 37 x K B 23, Body of alveolar crest group of fibres.
H K 13 x K B 43, Haversian canal.

2. The horizontal group, consisting of those fibres which pass out at right angles to the long axis of the tooth and are attached to the alveolar process a little below its crest.

3. The oblique group, consisting of those fibres which pass from the cementum in an oblique direction occlusally, and are attached to the alveolar process. These fibres constitute the body of the pericementum and cover the main body of the root of the tooth (figs. 18 and 19).

4. The apical group, consisting of those fibres which are attached about the apex of the tooth and extend in bundles to the surrounding alveolar process (figs. 20 and 21).

2. *Fibroblasts.*

Found everywhere mixed among the principal fibres, there are connective-tissue cells whose cytoplasm is extended into processes, which, as a general rule, run in a direction parallel with the root. The cells are stellate or spindle-shaped and their fibres act as supporting structures to the arteries, veins and nerves.

3. *Cementoblasts.*

These cells are really specialized osteoblasts and lie upon the cementum of the root. Their function is to form cementum in the

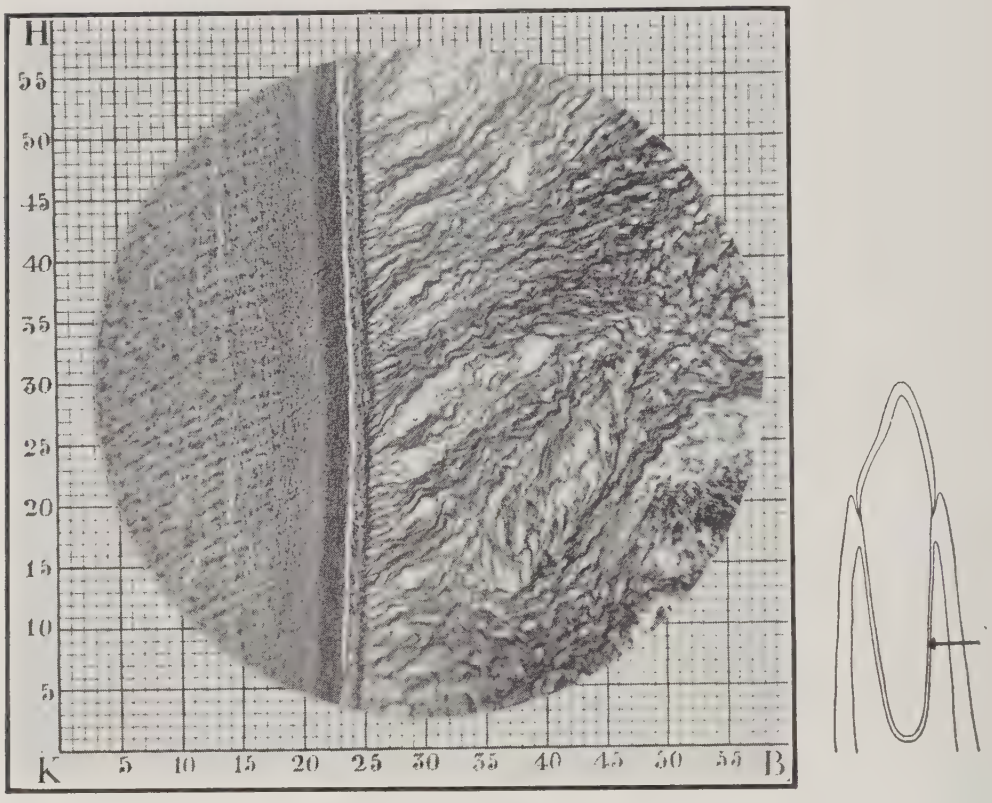


Fig. 18— x 187. Oblique group of principal fibres of the pericementum Note that the fibres take a course nearly parallel to each other as they pass out from the tooth surface.

H K 25 x K B 25, Insertion of fibres into the cementum.
H K 23 x K B 30 } Spaces formerly occupied by epithelial cells of
H K 20 x K B 30 } the pericementum.

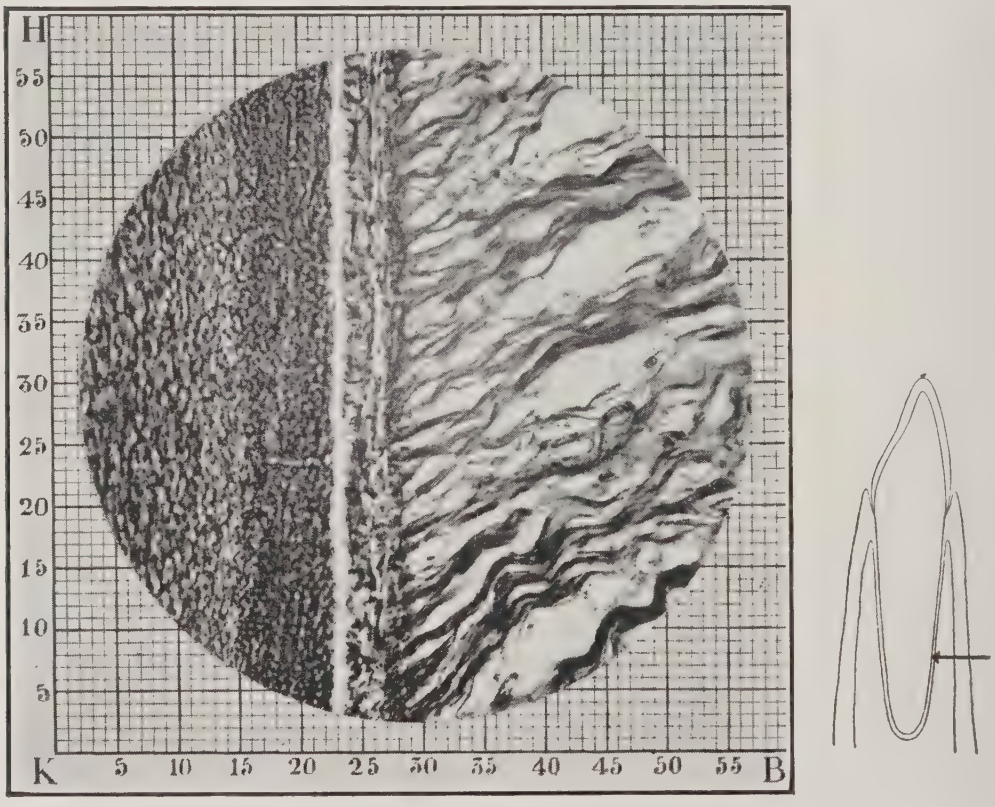


Fig. 19— x 750. Higher magnification of certain fibres belonging to the oblique group.

H K 25 x K B 23 to H K 25 x K B 28. Width of cementum.

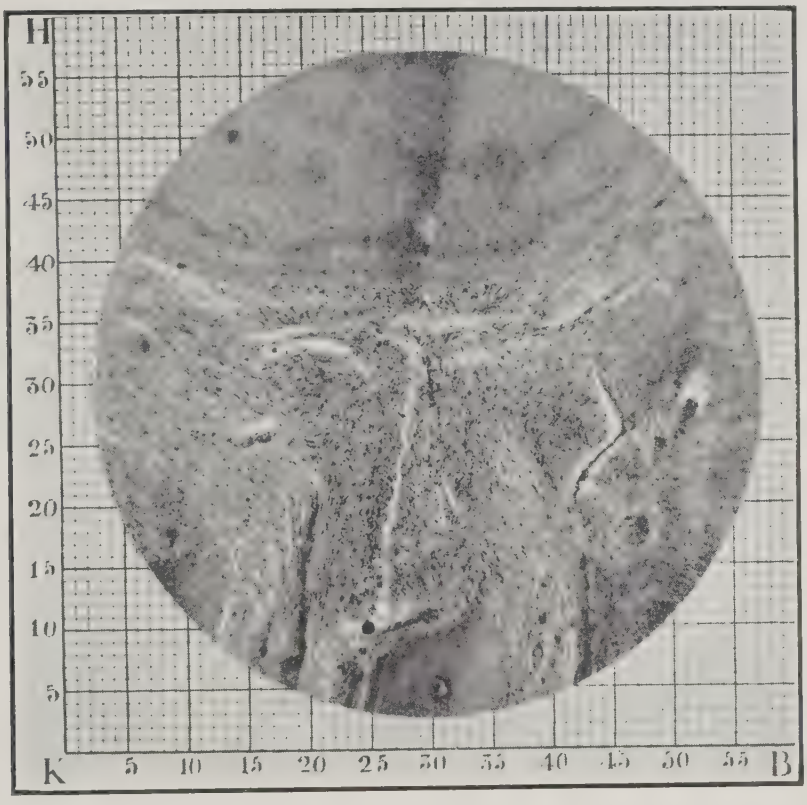
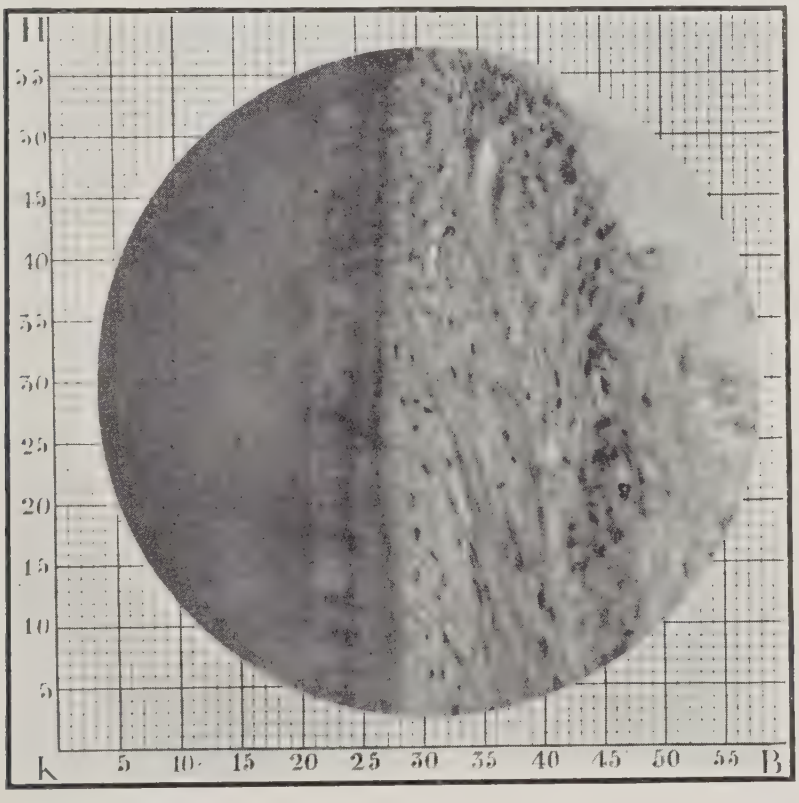


Fig. 20— x 94. Apical group of principal fibres of the pericementum. These fibres pass out from the apical cementum in all directions.
H K 38 x K B 29, Lower border of the apical cementum.
H K 23 x K B 30, Body of apical group.
H K 9 x K B 32, Alveolar bone.



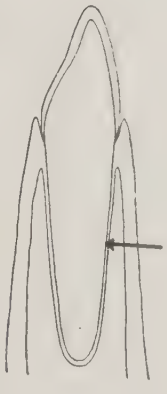


Fig. 21. Method of insertion into the cementum of the principal fibres of the gingivae and pericementum.
H K 26 x K B 27, Fibres passing into the cementum.
H K 25 x K B 20, Dentinal-cemental junction.

same manner as osteoblasts lay down the calcium salts in the building of bone. Noyes states: "They are always flattened cells, sometimes almost scale-like, and when seen from above, very irregular in outline. This irregularity in outline is due to the projections of the cytoplasm around the fibres as they spring from the cementum, the edges of the cell being notched and scalloped to fit about them. There is a central mass of granular cytoplasm which contains an oval and more or less flattened nucleus from which the cytoplasm extends in projections, passing partly around the fibres." Certain cementoblasts here and there become enclosed in the forming matrix as cement corpuscles, lying in stellate lacunae (fig. 22).

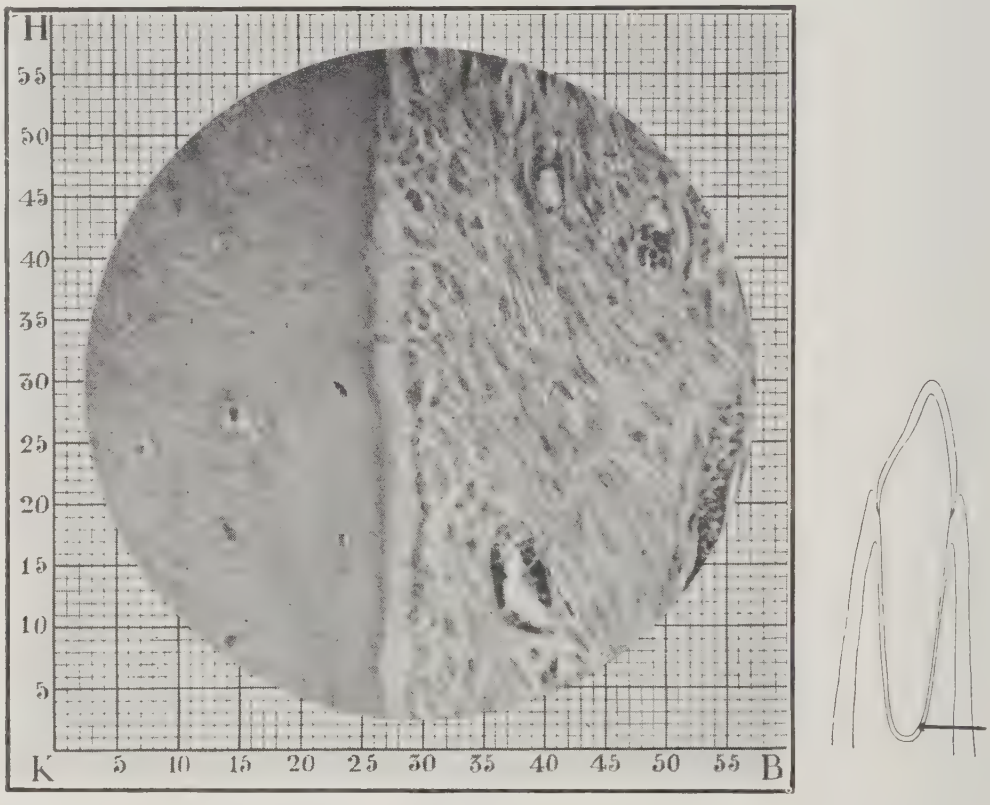


Fig. 22— x 500. Cementoblasts.

H K 30 x K B 29, Cementoblasts; these cells are shown in a vertical column on the surface of the cementum.

H K 28 x K B 15, Cement corpuscle lying in its lacuna.

H K 15 x K B 38, Blood-vessel in pericementum.

4. *Osteoblasts.*

The osteoblast is the embryonic or "free form" of the bone-cell and once formed, is capable of marked activity. They have the power of producing a matrix which becomes calcified. They also play a part in the absorption of bone. The osteoblasts of the pericementum do not differ from osteoblasts found elsewhere in the body. In children and adolescents they are present in large numbers in the pericementum, on the inner surface of the alveolar process, between the principal fibres. But in normal adult life they are found only

at irregular intervals. The presence of osteoblasts in great numbers on the surface of the bone is due simply to the proliferation of the osteoblasts which normally lie on the surface of the bone and in the mouths of the Haversian canals (fig. 23).

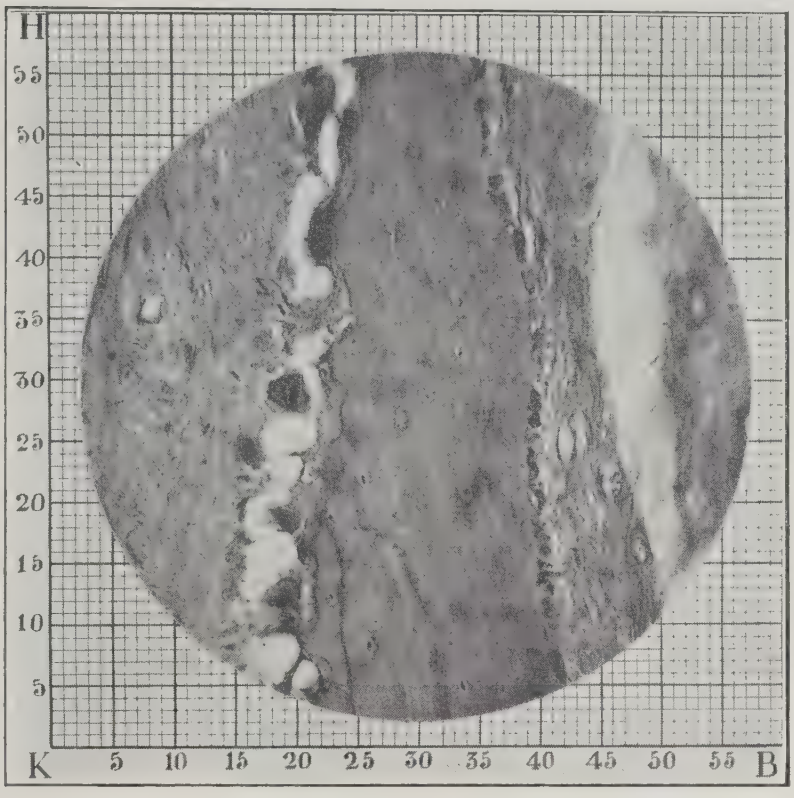


Fig. 23— x 375. Osteoblasts shown on the surface of the alveolar bone in the bifurcation of a molar.

H K 25 x K B 30, Alveolar bone.
H K 29 x K B 40, Layer of osteoblasts on surface of alveolar bone.
H K 30 x K B 20, Multinucleated osteoclast.
H K 30 x K B 10 } Pericementum.
H K 30 x K B 45 }

5. *Osteoclasts.*

Osteoclasts are found occasionally upon the surface of the alveolar wall and the cementum. They do not differ from the osteoclasts found elsewhere in bone. Foreign-body giant-cells are formed by the fusion of new-formed cells derived from the connective-tissue cells or from old endothelium. Arey contends that the chief source of osteoclasts, however, is from old osteoblasts and bone cells. They are large multinuclear cells which seem to be capable of absorbing bone, their cytoplasm lying in contact with the surface on which it acts (fig. 24).

6. *Epithelial Structures.*

Lying close to the cementum, but not in actual contact with it, are structures made up of epithelial cells in the form of strings or loops. The epithelium on the surface projects down into the connective-tissue stroma and a short distance from where they end, these cords of

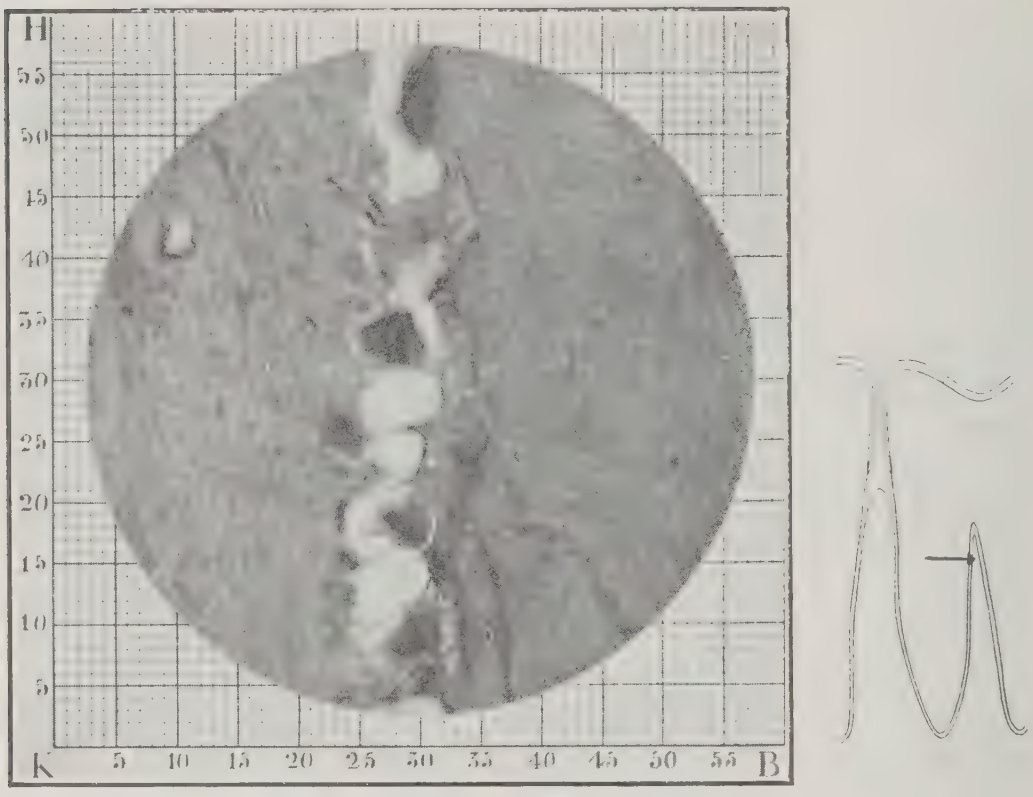


Fig. 24— x 450. Multinucleated osteoclast on the surface of the alveolar bone. Higher magnification of the osteoclast shown in preceding figure.
H K 35 x K B 28, Multinucleated osteoclast. Note numerous nuclei.

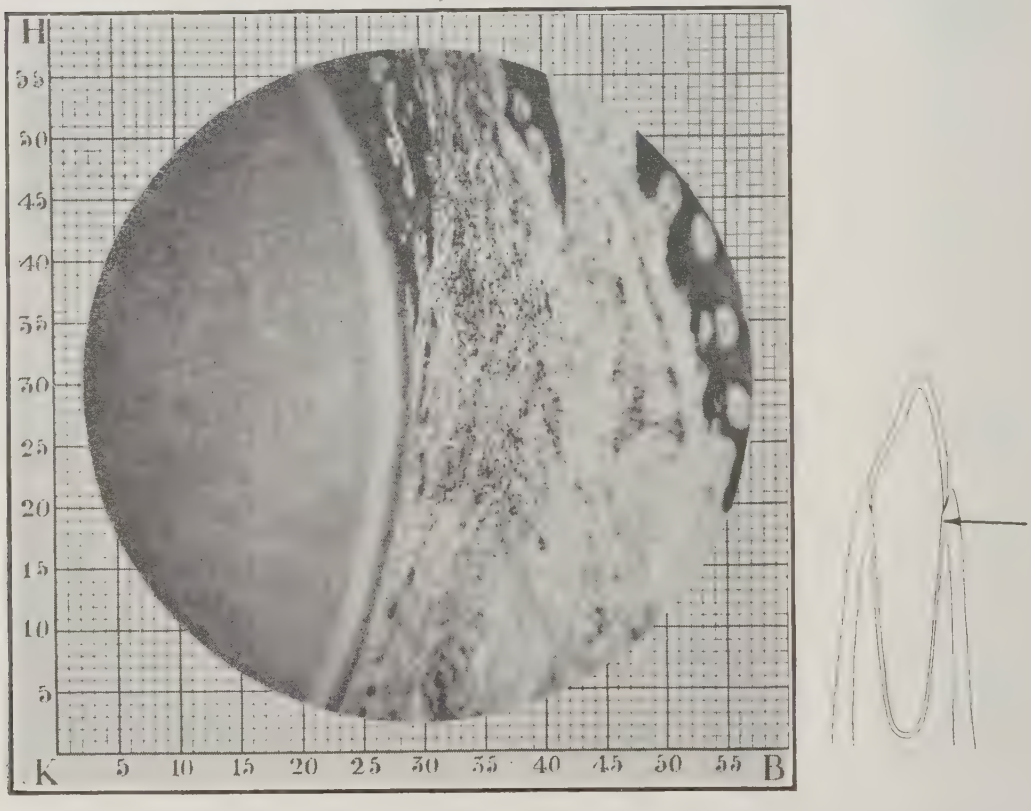


Fig. 25— x 125. Cords of epithelial cells in the pericementum. Cross section.
H K 31 x K B 30 }
H K 23 x K B 30 } Epithelial cords.
H K 15 x K B 29 }
H K 20 x K B 26, Surface of cementum.
H K 20 x K B 33, Pericementum.

epithelial cells appear, and in many instances pass around the entire root of the tooth (figs. 25 and 26).

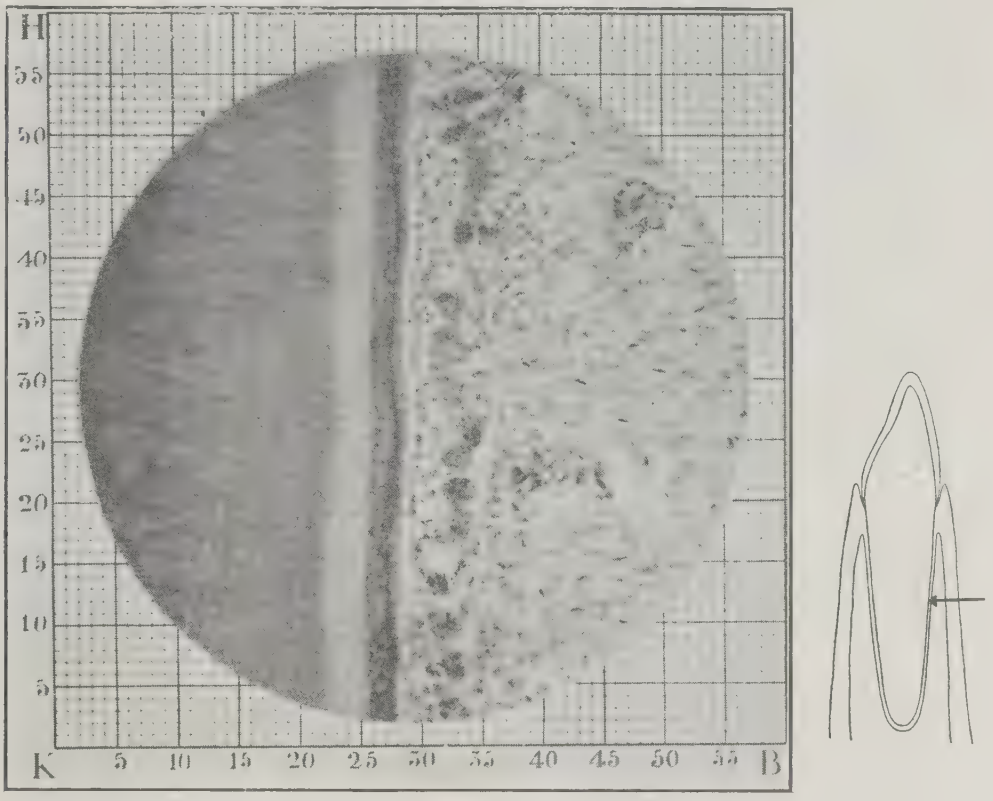


Fig. 26— x 250. Cords of epithelial cells in the pericementum. Longitudinal section.

H K 47 x K B 34 } Epithelial cords.
H K 30 x K B 33 }
H K 29 x K B 30, Cementoblasts.
H K 30 x K B 27, Cementum.
H K 16 x K B 32, Small blood-vessel in pericementum.

7. *Blood-vessels.*

The pericementum has an abundant blood supply. The vessels enter the periapical tissues where they branch and pass into the pericemental tissue, running parallel with the long axis of the root (fig. 27). Some of the branches pass through the apical foramen to nourish the pulp. Sometimes as many as eight or ten branches enter the body of the pericementum and passing occlusally, give off branches and anastomose with other vessels through the Haversian canals in the process. At the crest of the alveolar prosess, anastomosis occurs with the vessels of the gingivae, periosteum and alveolar mucosa.

8. *Nerves.*

The nerves of the pericementum follow the course of the blood-vessels. Ten to twenty-five medullated fibres enter the periapical tissues with the blood-vessels, some entering the pulp, others passing upward through the bone and form with the others a plexus from which filaments are given off. These terminate in free endings in the pericementum (figs. 28 and 29).

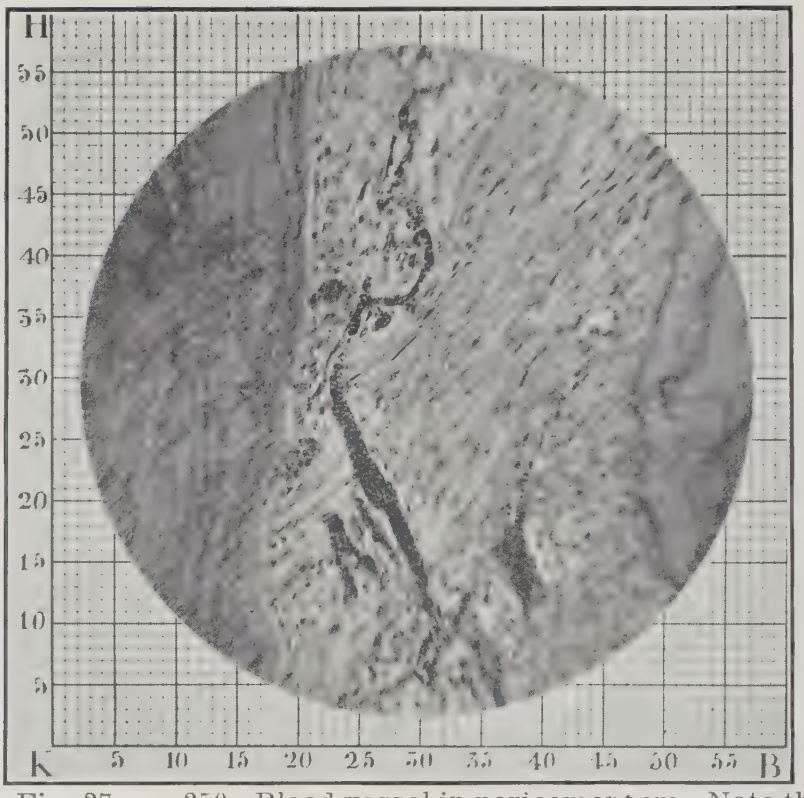
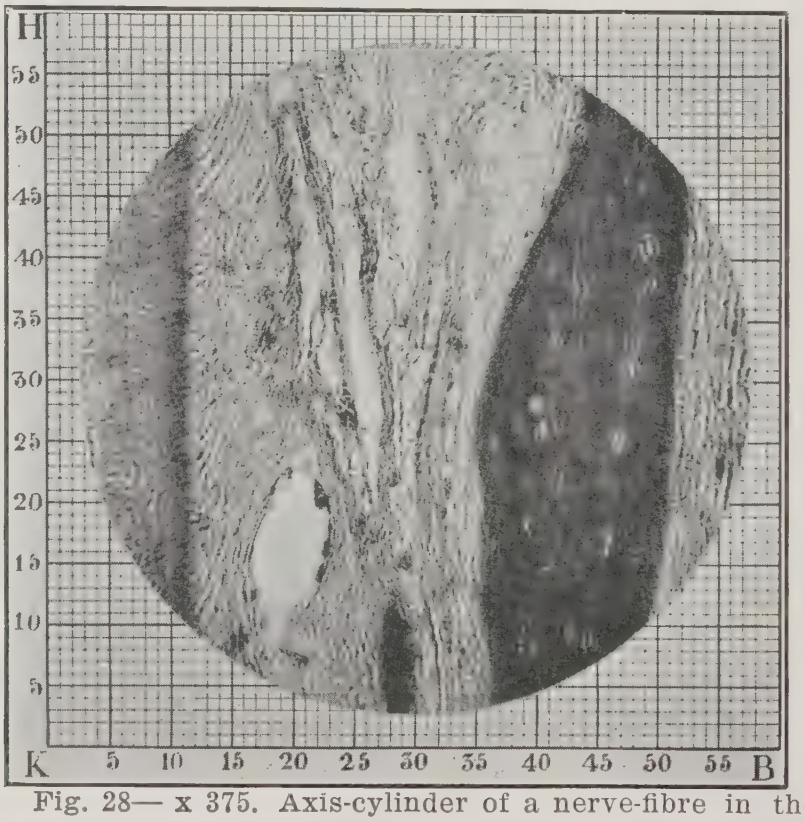


Fig. 27— x 250. Blood-vessel in pericementum. Note the unusual extent of the vessel shown on one plane.

H K 22 x K B 26, Blood-vessel at largest diameter.
H K 39 x K B 30, Branch given off from larger blood-vessel.
H K 31 x K B 21, Cementum border.
H K 31 x K B 47, Border of alveolar bone. The distance from K B 21 to K B 47 indicates the thickness of the pericementum at this point.
H K 30 x K B 35, Bundles of fibres in oblique group.



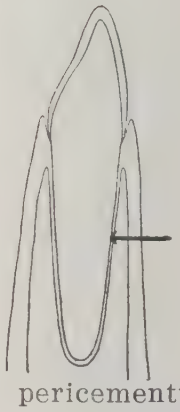


Fig. 28— x 375. Axis-cylinder of a nerve-fibre in the pericementum.

H K 32 x K B 31, Small axis-cylinder of pericemental nerve, passing longitudinally.
H K 30 x K B 11, Cementum border.
H K 30 x K B 35, Border of alveolar bone.
H K 17 x K B 21, Cross section of large blood-vessel.

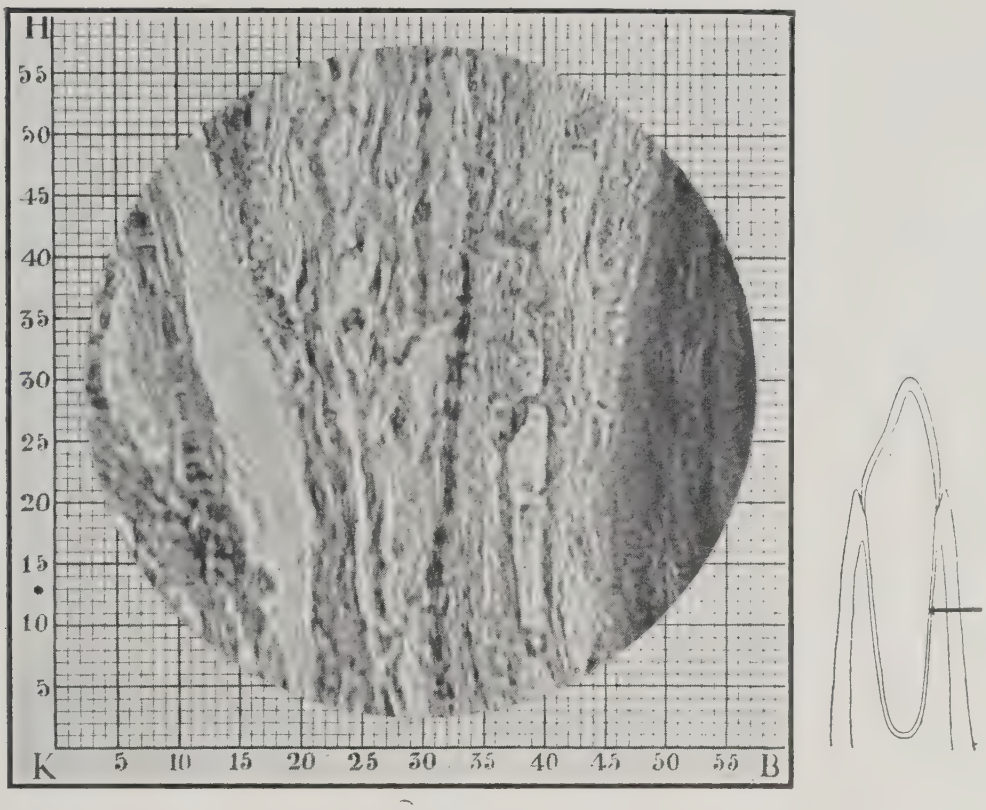


Fig. 29— x 1125. Higher magnification of the axis-cylinder of a pericemental nerve-fibre.

H K 41 x K B 33 to H K 4 x K B 32, Axis-cylinder of pericemental nerve.

THE ALVEOLAR PROCESS.

Since the bone which supports the teeth is continuous, both in its gross and minute anatomy, with the bone which makes up the body of the maxillae, it has been found difficult to define the *alveolar process*. It may perhaps be acceptably described as the bone which immediately invests the roots of the teeth together with such adjacent bone as is intimately concerned in their support.

Many efforts have been made in the past to show that the alveolar process is made up of bone having some peculiar characteristics which differentiate it from other osseous tissue in the body. We have been unable to find any histologic evidence to warrant this assumption. It has a specialized function, and because of its function and its location, it is peculiarly subject to disease. This fact does not, however, justify the assumption that loss of the alveolar process is an inevitable phenomenon.

Bones are formed either by ossification of connective-tissue membranes or by the replacement of cartilage by osseous tissue. They nearly always develop from a connective-tissue foundation, even where they are laid down in places formerly occupied by cartilage. Membranous bones seem to be produced by a change in the function of certain cells which acquire the property of laying down an intercellular, formless ground-substance in which they bury them-

selves. This later becomes calcified. These cells are known as osteoblasts. The osteoblasts form for themselves, in the hyaline ground-substance they have produced, little cavities in which they lie, their numerous tiny branches extending out in every direction, into small canaliculi. The osteoblasts thus become stationary and are termed "bone-cells." At this stage, the tissue is known as osteoid tissue. In a short time, there takes place, a deposition of calcium throughout, and the tissues become hard bone.

Investigators are not unanimous in their views as to the manner in which the calcium salts are laid down. The process consists of a deposition, in the organic ground-substance, of very fine granules, which fuse together to form a homogeneous mass. Watt, of the University of Toronto, has observed these granules only at the extreme advancing edge of new bone. These granules might represent the product of a process of either precipitation or secretion. Three main conceptions have been put forward to explain the deposition of the lime salts in bone. According to Watt, these may be summed up as follows:—

1. The salts are deposited by precipitation in the matrix of a soluble salt of calcium, such as the chloride, by carbonates and phosphates found in the tissues.

2. A modified precipitation theory. According to this theory a soluble double-salt, calcium carbonophosphate, is carried in the blood and deposited in the bone by change of carbon dioxide concentration in this tissue. This precipitates the double salt, which then breaks up into calcium carbonate and di-calcium phosphate, which latter is immediately converted into the tri-calcium phosphate.

3. The secretion theory. This explains the presence of these salts as due to cellular activities, calcium being taken from the blood, and combined with the carbonate radical in the cell, or with the phosphate radical, this latter being derived from the nucleus, either as a normal cell product or as a product of degeneration.

In a recent work on the deposition of bone salts, Watt has expressed his views as follows:—"The most logical complete explanation of the deposition of bone-salts, it seems to me, is to be obtained by combining two of the above views. Evidence is in favor of the calcium being carried in the blood as the soluble double salt, calcium carbonophosphate, which supplies to the bone, both carbonate and phosphate in exactly the proper proportion. This salt is taken from the blood, and deposited in the bone by the action of the osteoblasts. This view also provides for a reversibility of direction of travel of the calcium. The cell may take up the bone-salts and pass them back into the blood, a process which is well known and recognized in bone absorption."

Macewen holds the view that the bone-cell has the function of surrounding itself with a calcareous zone at the period when the osteoblast reaches maturity and becomes stationary in its gound-substance.

There is abundant evidence that the osteoblasts have the power of absorbing bone. Nothing could be more convincing in this relation, than the investigations of Gallie and Robertson of Toronto, in the repair of bone. They have shown, in their numerous experiments with grafts of bone, which are absorbed and replaced by new osseous tissue, that this process of absorption is almost solely dependent upon the activity of the osteoblasts.

PART II: GINGIVITIS.

Gingivitis may be defined as the re-active phenomena of the gingival tissues to any injury done to them.

Anything that is capable of injuring the gingival tissues may produce in them the phenomena of inflammation. The essential thing about the harmful agent always is, that it injures the tissues, as inflammation takes place because tissue has been injured. The reaction of the tissues to any injurious agent and to the injury produced by it, differs according to the nature of that agent and to the severity of the injury. The function of that reaction is to eliminate the noxious agent, to counteract its action and to repair the injury produced.

Mallory states:—"The reaction may be evidenced chemically by metabolic changes, such as alteration of secretion and excretion: morphologically, by the presence of serum, fibrin, and leukocytes and by the proliferation of cells; and physiologically, by alteration of functional activity." Again he states:—"The morphologic changes which take place as the result of the action of various injurious agents vary greatly, depending on the strength and character of the latter." Also: "In general we may divide the injurious agents into two groups, the strong and the mild irritants (with all gradations between) although it is difficult to confine some of them exclusively to either group."

Due to the epoch-making discoveries of Virchow, Cohnheim, Marchand, and others, our conception of the changes which take place in the tissues as the resultant of the action of strong injurious agents, has been clarified, and it is generally acknowledged that acute inflammation is anatomically manifested both macroscopically and microscopically by three main processes. Mallory has described them as follows:—

" 1. Circulatory disturbances.
2. Inflammatory exudation.
   (a) Exudation of lymph (including formation of fibrin).
   (b) Emigration of leukocytes (chiefly of the polymorphonuclear leukocytes).

3. Proliferation of emigrated endothelial leukocytes and lymphocytes, of fibroblasts, of vascular endothelium and of epithelial cells, if included in the lesion."

A detailed description of the phenomena of acute inflammation, is a more or less laborious process and one which shall not be attempted in this paper. As these changes induce certain gross anatomical appearances, which, as a rule, can be distinguished clinically, and are known as the classic signs of acute inflammation, namely, redness, heat, swelling and pain, it is advised that students of periodontal pathology should study these phenomena through the medium of a standard text-book of general pathology.

The reaction of the tissues to *mild* injurious agents from a histopathological point of view is of equal importance to the reaction to *strong* irritants, manifested in acute inflammation. Its significance in dental pathology has not been been fully appreciated. The *mild* injurious agents include micro-organisms which produce weak toxins, and also strong toxins from different sources which have been so diluted as to act as mild irritants. From the standpoint of focal infection, the greatest problem in the dental profession to-day, and from our present viewpoint as well, the inflammatory reaction to these mild irritants is of much greater moment than the reaction to *strong* injurious agents.

The lack of appreciation, pathologically, of the relation of these reactions to those produced by the strong irritants, and the fact that when an injurious agent acts mildly, one or all of the classic signs of acute inflammation may be lacking, account largely for the obscurity surrounding this phase of the subject.

According to Mallory, "The most common inflammatory reaction to mild irritants consists of an exudation and proliferation of endothelial leukocytes which sometimes accumulate in very large numbers. To the endothelial leukocytes may be added serum (from which fibrin may form) lymphocytes, polymorphonuclear leukocytes and rarely eosinophiles."

Again,—"Other injurious agents attract lymphocytes in large numbers so that focal accumulations of some size may occur in various organs and tissues: for instance in the heart, kidney, adrenal and skin."

The gingivae are frequently the site of inflammatory processes, which *often* persist over a considerable period of time. These manifestations of the defensive processes of the gingival tissues are revealed only to the competent observer, his ability to interpret them depending upon his understanding of the fundamental principles of inflammation, and of the histology of the tissues involved. The phenomena of *acute* inflammation are *displayed* in the gingival tissues as variegated clinical pictures; but the *inflammatory* reactions to mild injurious agents are *generally unapparent* and are detected with difficulty.

It must be kept in mind that bacterial invasion of the gingivae rarely takes place without a pre-existing state of local lowered resistance. Careful investigations have proved that from a bacteriological standpoint, the causative factors of these infections are common to all mouths. All factors in the disturbance of capillary circulation can be regarded as productive of lowered resistance. Accumulations of foreign material, restorative impingement, food impaction, incorrect mouth toilet, etc., in this relation are so well known as to need no further comment.

During a period in which the alveolar crest may be under great strain in its effort to maintain a balance of the forces acting against it, as in traumatic occlusion, and for the period immediately following disturbance of that balance, the marginal gingivae in many instances presents clinically a characteristic hyperemia. This is to be regarded as a sign of pericemental trauma. Upon establishment of equilibrium of the forces, by readjustment of mechanical factors that to a considerable degree are under the control of the operator, namely, the point of application and the direction of the initial force, these tissues as a rule, quickly regain their normal tone and color. When the tooth is pushed against the alveolar crest and associated alveolar wall, an abnormal condensation of pericemental tissue lying between the tooth and these areas takes place. Dependent upon the intensity of this condensation, the blood-vessels are more or less constricted and the flow of blood through them is partly cut off. When the pressure is removed there is a consequent dilatation of the blood-vessels. This condition is reflected in the terminal capillary loops in the marginal gingivae, a clinically observable disturbance.

Inflammations of the gingivae may be classified as follows:—

1. Marginal gingiva.
   - A. Acute { Non-Ulcerative. / Ulcerative (fuso-spirillary).
   - B. Sub-acute.
   - C. Chronic { Non-Ulcerative. / Hyperplastic / Ulcerative (fuso-spirillary). / (Crevicular). / (Calcular).
2. Cemental Gingiva.
   - A. Acute { Non-Ulcerative. / Ulcerative (fuso-spirillary).
   - B. Sub-acute.
   - C. Chronic { Non-Ulcerative. / Ulcerative.

It is not our intention to give here a detailed description of these thirteen types of gingivitis. Nevertheless a brief description of some of the more salient ones is a prerequisite in the understanding of some of the more complicated periodontal conditions to be described later.

1. *Marginal gingiva.*

ACUTE FUSO-SPIRILLARY GINGIVITIS.

Regarding this form of gingival disease, Stillman and McCall have stated:—"The chief characteristics are:—sudden onset; a sensitive and serpiginous ulcer, consisting of necrotic tissue elements and a creamy pasty exudate; characteristic fetor of the breath; hemorrhages, either spontaneous or induced by touch; a neurosis exhibiting malaise, mental depression, insomnia, restlessness; loss of appetite; profuse flow of saliva, often with a metallic taste; temperature above normal."

The following photomicrographs were made from sections taken from the gingivae in a case of this disease which occurred about the upper left central, lateral, and cuspid. Upon microscopic examination, it is observed that most of the epithelial cells have been cast off, and many of those present are swollen and filled with drops of exudate which crowd the nuclei against the cell-body. The exudation consists of serum and leukocytes; fibrin has been deposited as a layer of interlaced fibrils and mesh works matted together entangling leuko-

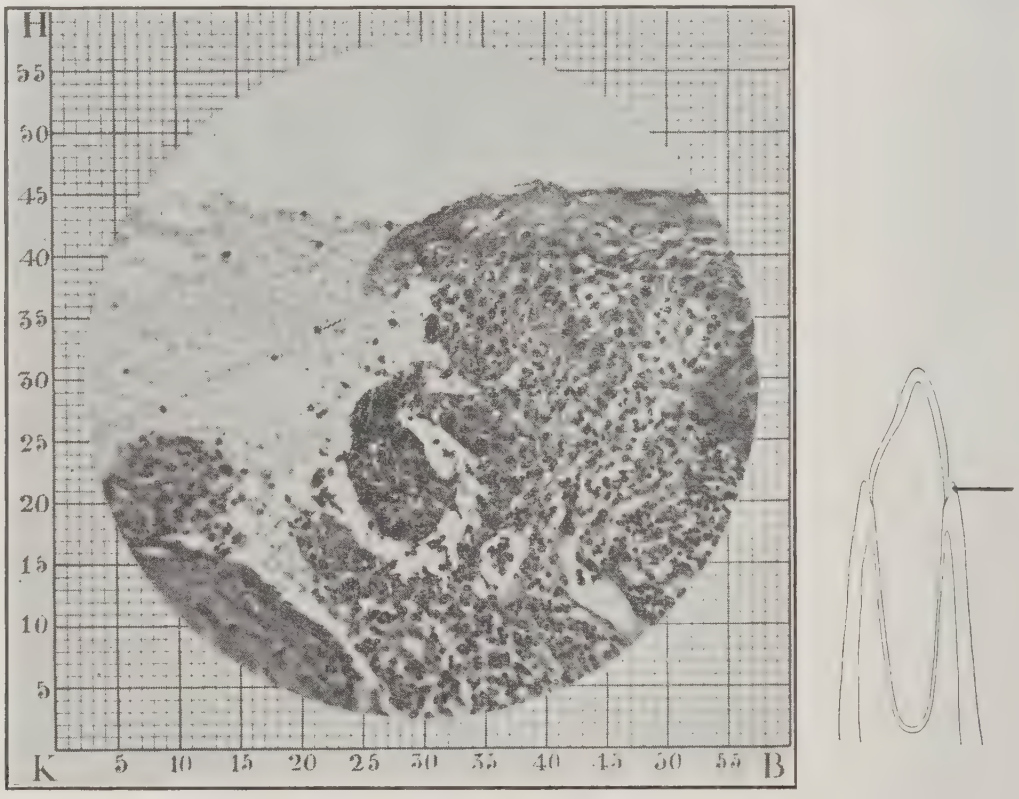


Fig. 30— x 280. Acute Fuso-Spirillary Gingivitis. Specimen taken from the marginal gingiva. Much epithelium has been lost and replaced in part by a fibrinous exudate.

The area shown as being bounded approximately by the lines within the points, H K 45 to H K 25, and K B 28 to K B 25, has lost its epithelium and some underlying connective tissue, and now shows a faintly stained fibrinous exudate.

H K 43 x K B 37, Epithelium as yet intact.

H K 35 x K B 52, Connective-tissue papilla, showing leukocytic infiltration.

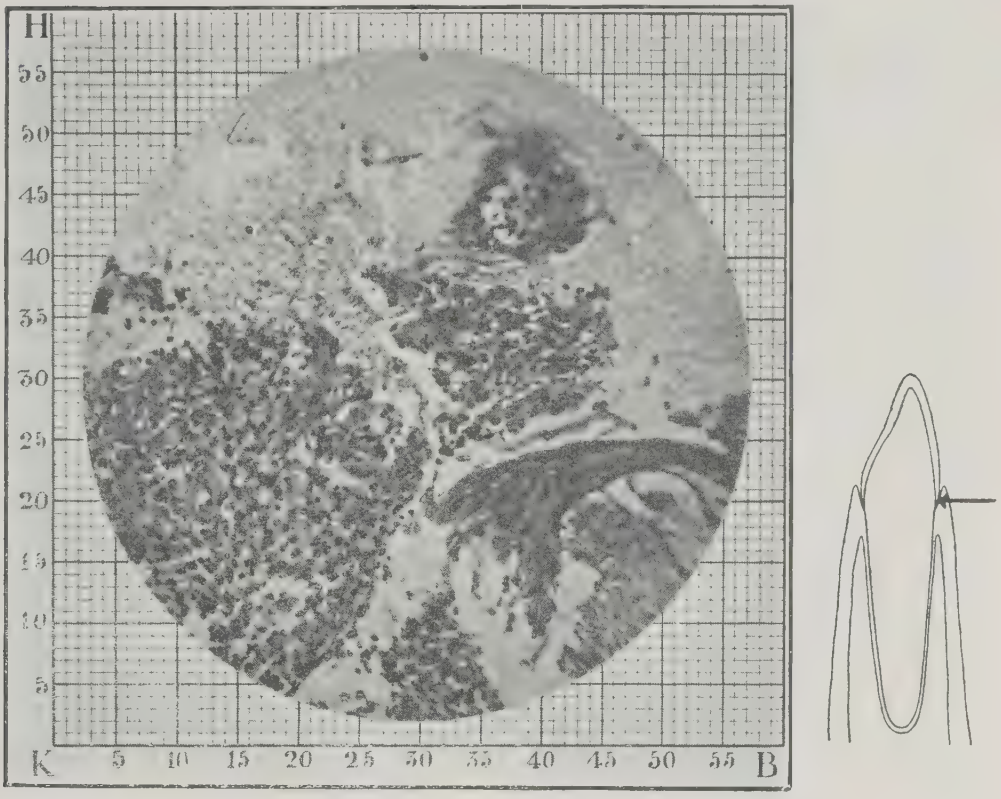


Fig. 31— x 280. Acute Fuso-Spirillary Gingivitis. Specimen removed from the marginal gingiva. The fibrinous exudate which has replaced part of the lost gingival tissue is shown in the upper part of the photomicrograph. The remaining tissue exhibits pronounced leukocytic infiltration.

cytes and bacteria. Red blood-cells have escaped from the blood-vessels and are found free in the surrounding mesh-work (figs. 30 and 31).

CHRONIC NON-ULCERATIVE.

From a microscopic examination of marginal gingivae showing long standing inflammatory reaction, the following has been noted. There is a large infiltration of leukocytes in the connective-tissue stroma beneath the surface epithelium of the marginal gingivae. These cells lie between the bundles of fibres and in some instances are present in the papillae. The blood-vessels are dilated and filled with red blood-cells. A very marked feature is the predominance of plasma cells. Lymphocytes and eosinophiles are also present. The main function of the lymphocytes appears to be the neutralization or disposition of injurious substances which are being absorbed through the lymphatics. It is not known just what function the eosinophile has, but it is thought to be the counteraction of certain injurious chemical substances (fig. 32).

HYPERPLASTIC.

The mucous membrane appears to be unchanged as a general rule, although in some areas the epithelium seems to project into the

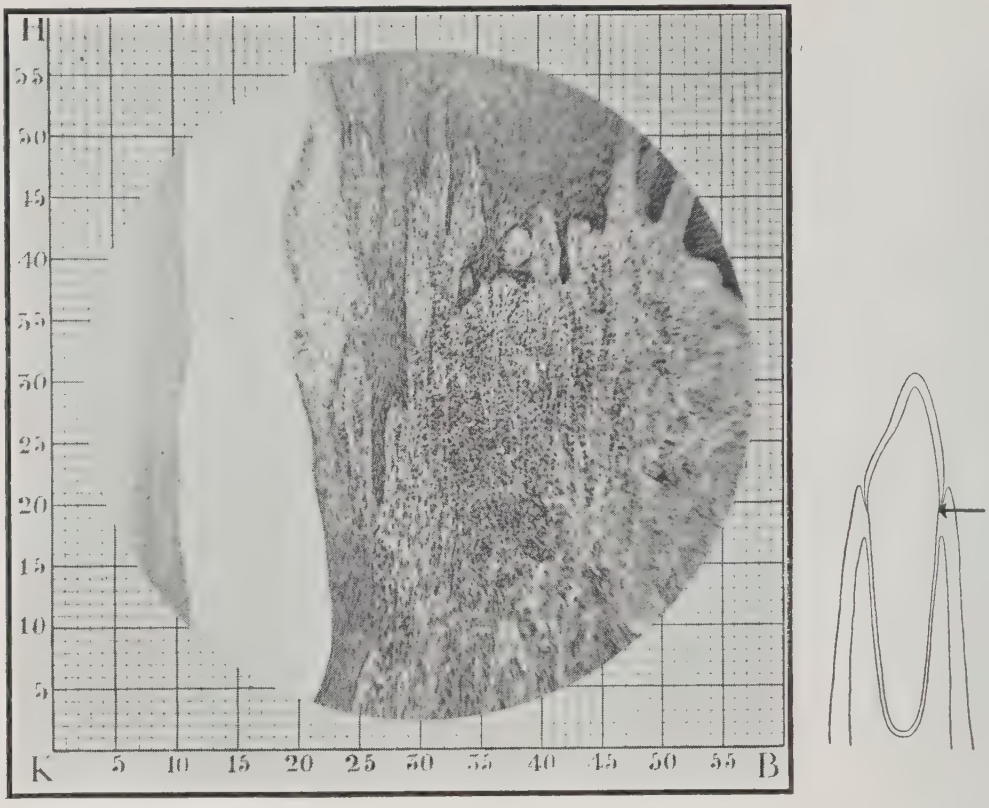


Fig. 32— x 125. Chronic Non-Ulcerative Gingivitis. This is the type of chronic gingivitis most commonly observed in the marginal gingiva.

H K 25 x K B 18, Gingival crevice.
H K 20 x K B 24, Crevicular epithelium.
H K 28 x K B 35, Pronounced infiltration by lymphocytes and plasma cells.
H K 50 x K B 40, Epithelium on external surface of marginal gingiva.

underlying tissue somewhat farther than normally. The bulk of the mass is made up of fibrous connective tissue, the bundles of which form trabeculae ramifying in all directions. Many of the blood-vessels are dilated. In the tissue-spaces around the blood-vessels and between the bundles of connective tissue, the cellular elements are very greatly multiplied. In the following slides, the majority of the cells are plasma cells, many of which contain droplets of hyalin (figs. 33 and 34).

CHRONIC ULCERATIVE (*Crevicular*).

The gingival crevice is normally cleansed by a serum which is derived from the marginal and cemental gingivae. When through growth of micro-organisms on the cervical enamel surface, the crevice becomes foul, especially when facilitated by a holding medium (such as etched enamel, that is, removal of the superficial cementing substance from between the enamel rods), necrosis of the crevicular epithelium takes place. By the death of epithelial cells, there is provided a suitable medium for the growth of micro-organisms and their penetration is facilitated. The resultant pathological condition is ulceration.

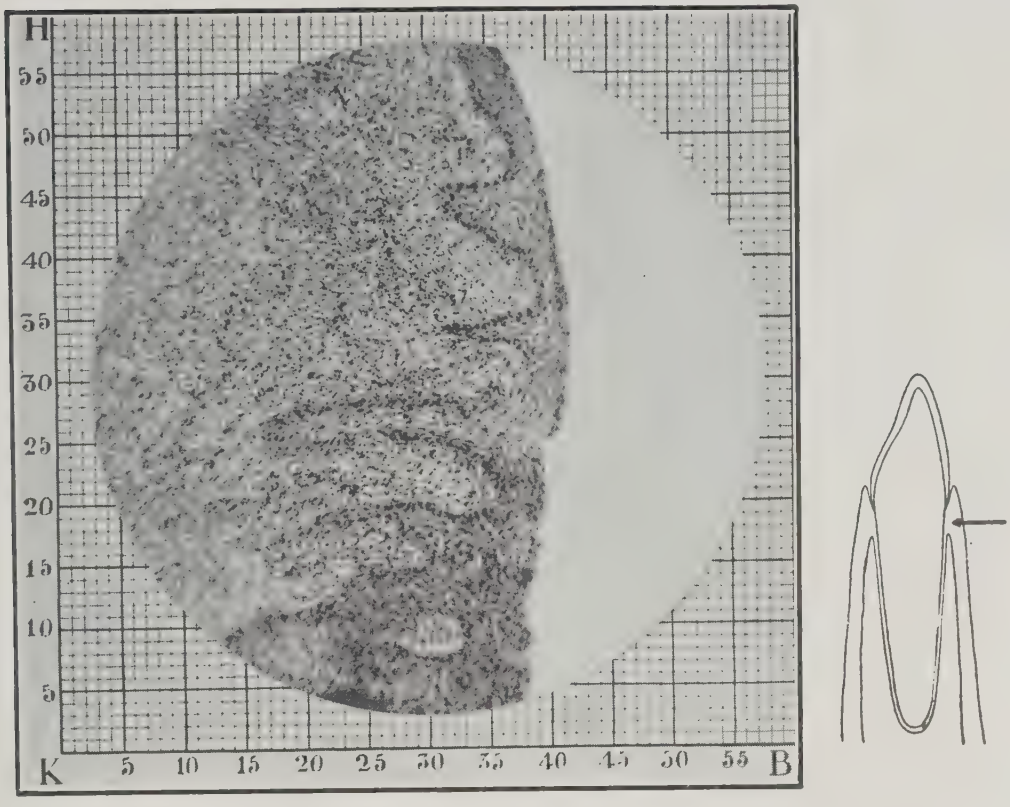


Fig. 33— x 94. Hyperplastic Gingivitis.

At H K 25 x K B 17 and H K 9 x K B 13, the epithelium appears to project into the underlying connective tissue farther than normal.

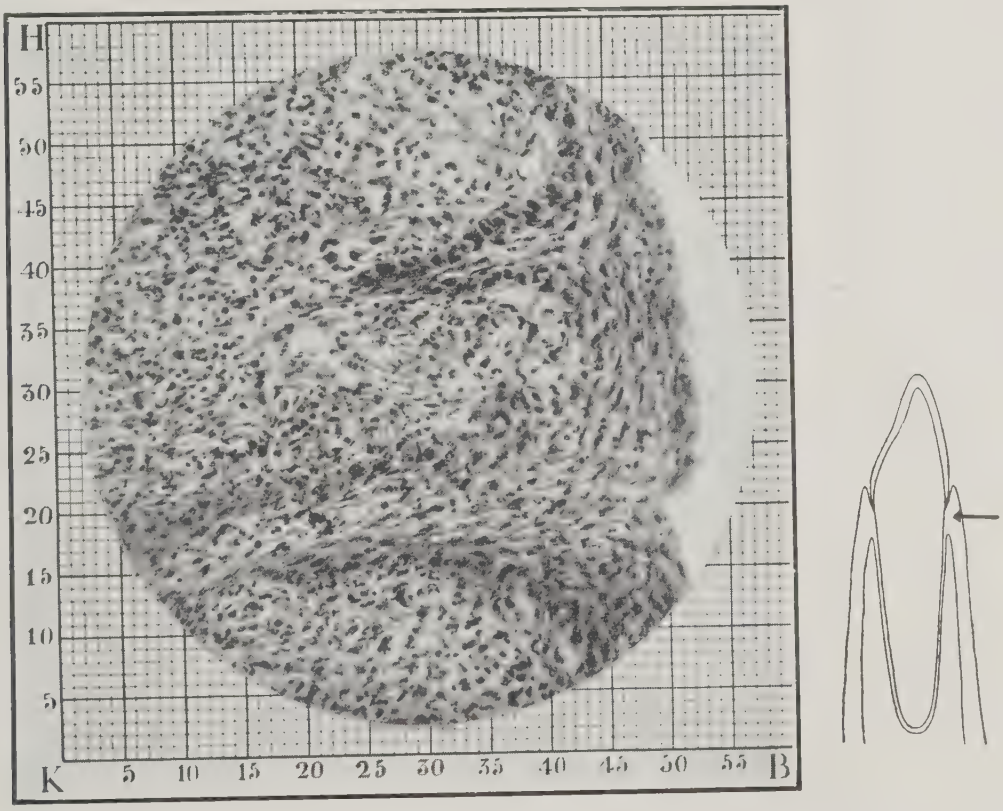


Fig. 34. Higher magnification of specimen shown in preceding illustration.

H K 17 x K B 9, Extremity of epithelial ingrowth.

Entire connective-tissue area shows an increase in its cellular elements and a marked leukocytic infiltration.

2. *Cemental Gingiva*

SUB-ACUTE.

This type of reaction in the cemental gingiva, in no way differs from that occurring in the marginal gingiva. The majority of these lesions are found at the base of the gingival crevice, where through imperfect junction of the epithelial cells with the cementum, or because of the extreme thinness of this defensive covering, the tissue underlying this area is not adequately protected. Plasma cells and lymphocytes are the predominating leukocytes.

We wish to more clearly distinguish these definite inflammatory reactions evidenced by such infiltration, from the normal condition existing in the gingival tissues in which slight collections of similar cells are constantly present.

A typical feature of the microscopic picture is the proliferation of the crevicular epithelium downward along the cementum. One of two things, as a rule, takes place. In the first place, by progression of the inflammatory process, ulceration is established and the proliferating epithelium is included in the loss of tissue. In the second instance, the epithelium, by proliferation, extends for some distance along the cemental surface. Then, separation from the cementum takes place with the resultant denudation of the cementum and the formation of a pocket. Infection of the tissue at the base of this pocket generally occurs, sooner or later, frequently with the establishment of ulceration (figs. 35 and 36).

CHRONIC NON-ULCERATIVE.

This type of inflammation in the cemental gingivae resembles that occurring in the marginal gingivae and described previously. The leukocytic infiltration, chiefly plasma cells and lymphocytes, is present between the bundles of fibres and occasionally in the papillae. In this type of lesion, there is to be found a tissue architecture which differentiates it from the sub-acute type, consisting in the presence not only of lymphocytes and plasma cells, but also of connective-tissue cells in various stages of development, from the fibroblast to the mature connective-tissue cell.

A suppurative gingivitis frequently follows this type, due to acute inflammatory changes in which the exudate passes through the epithelial covering to the pocket. A characteristic of this suppurative but non-ulcerative condition is the fact that the epithelium covering the denuded soft tissues remains intact. The epithelial cells are not lost until actual ulceration takes place.

Because of the mild character of the symptoms, this condition is readily overlooked in clinical examination. Because it is one of the early phases of a still more serious condition, we would stress the importance of its early detection and treatment (fig. 37).

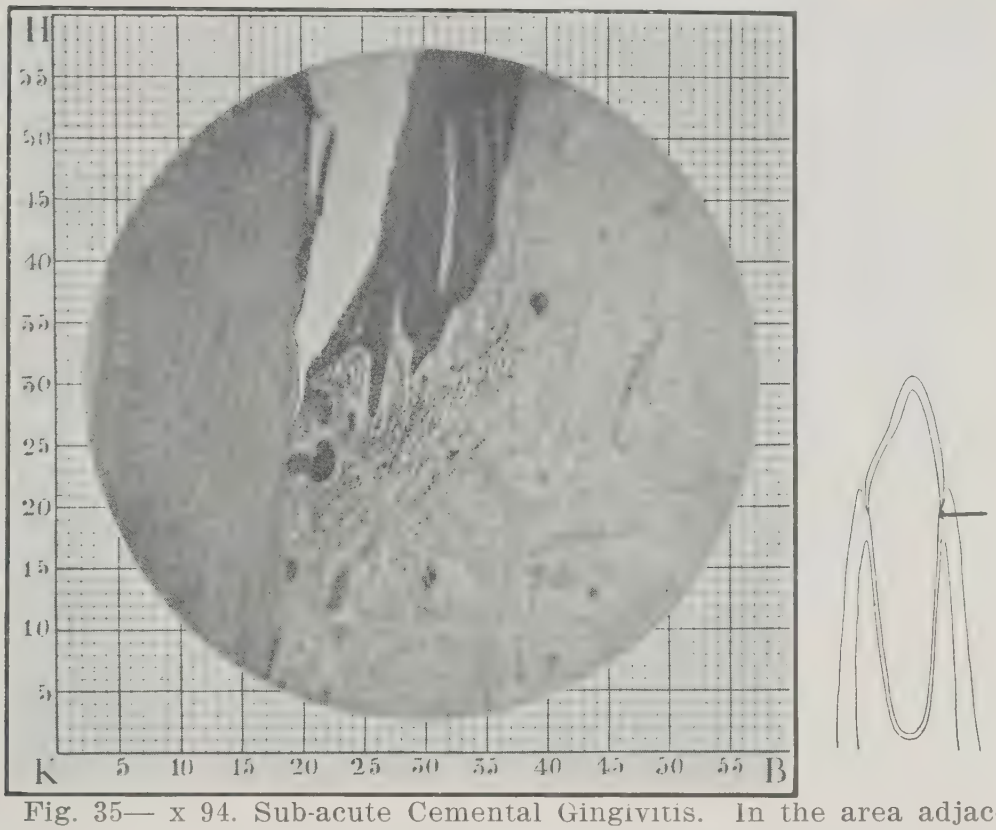


Fig. 35— x 94. Sub-acute Cemental Gingivitis. In the area adjacent to the base of the gingival crevice, there is to be seen a definite leukocytic infiltration (chiefly plasma cells and lymphocytes).

H K 29 x K B 20, Base of gingival crevice. Note imperfect junction of epithelium with cementum.

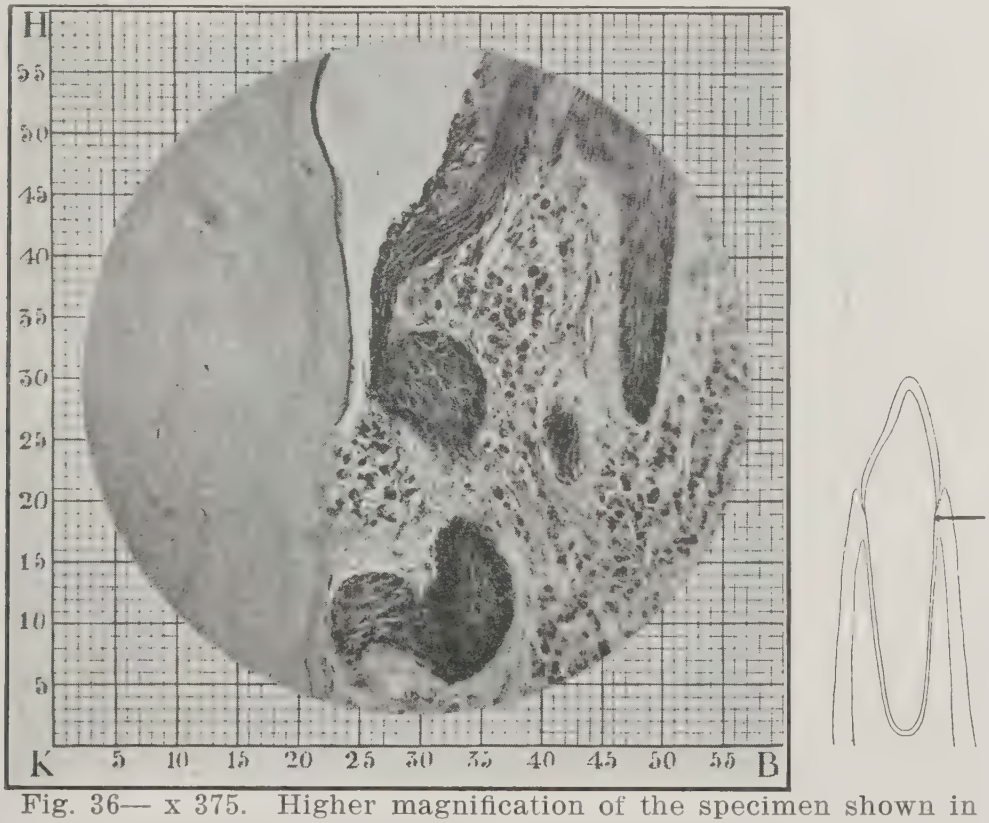


Fig. 36— x 375. Higher magnification of the specimen shown in the preceding illustration. The base of the gingival crevice.

H K 29 x K B 25, Base of gingival crevice; imperfect junction of epithelium with cementum.

H K 37 x K B 35
HK 22 x K B 27 Areas of marked leukocytic infiltration.

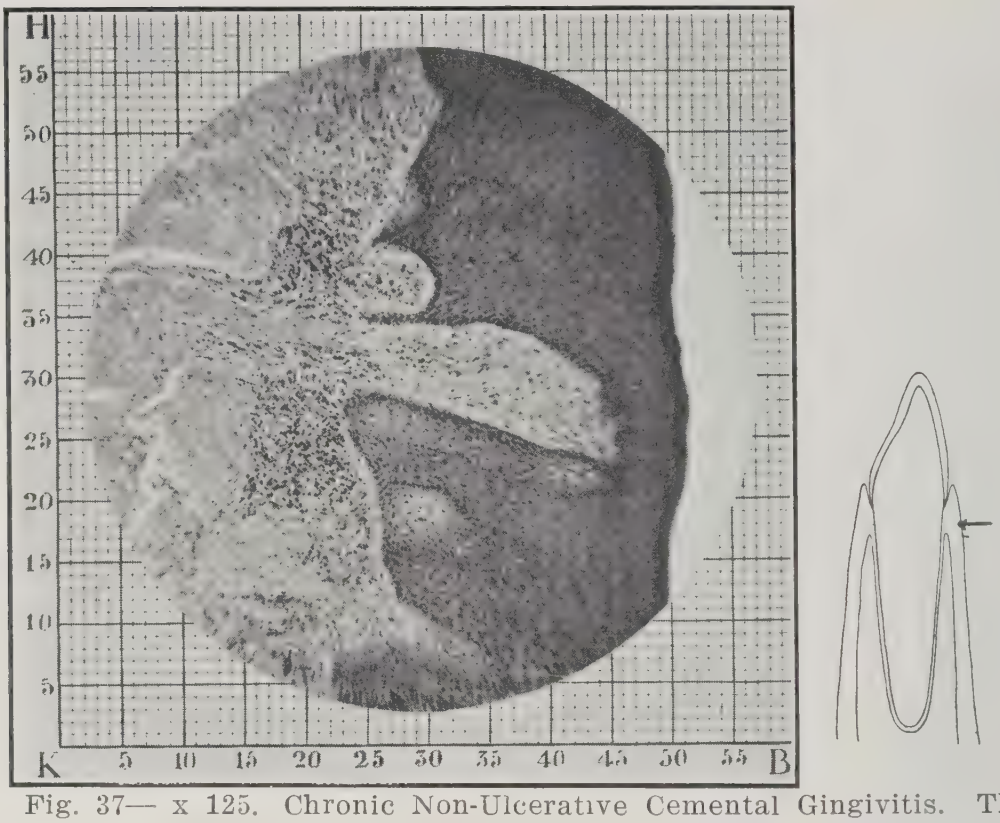


Fig. 37— x 125. Chronic Non-Ulcerative Cemental Gingivitis. This photomicrograph shows the external surface of the cemental gingiva. This is a parallel type to the chronic non-ulcerative marginal gingivitis. See Fig. 32.

H K 40 x K B 22 Areas of leukocytic infiltration, largely plasma cells
H K 25 x K B 20 and lymphocytes.
H K 30 x K B 40, Connective-tissue papilla.

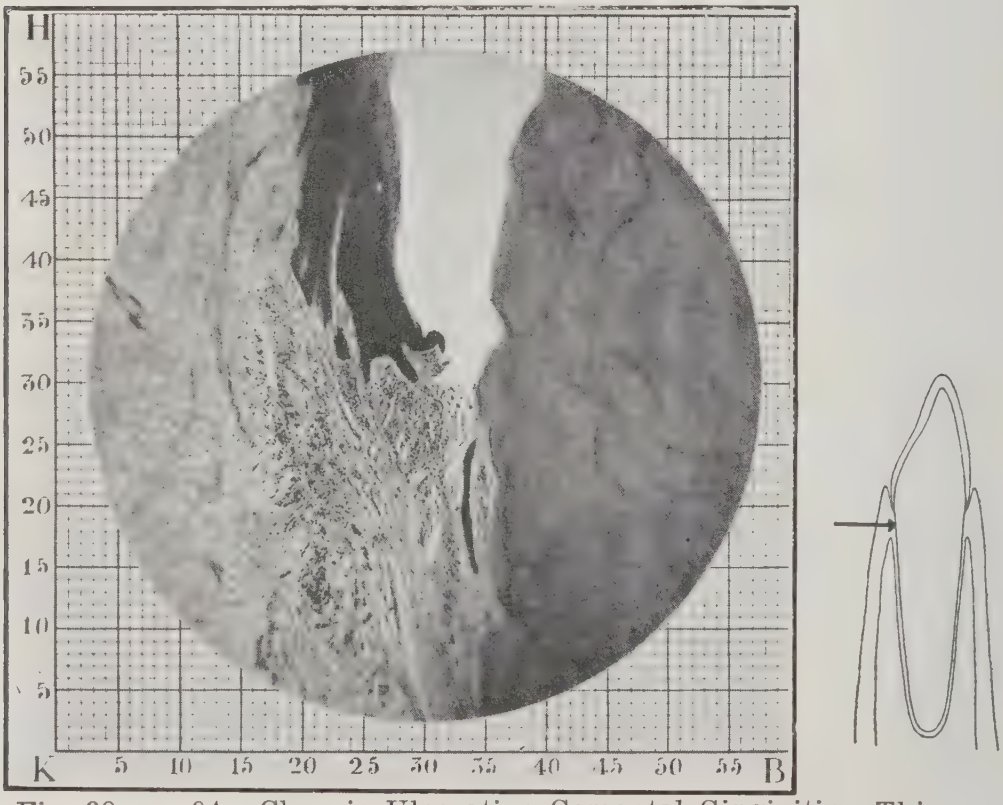


Fig. 38— x 94. Chronic Ulcerative Cemental Gingivitis. This specimen shows ulceration in the connective tissue at the base of the gingival crevice. Note the extensive infiltration in the adjacent tissue.

H K 30 x K B 33, Ulceration at the base of the gingival crevice. Marked leukocytic infiltration extending as far as H K 12 x K B 21.

H K 22 x K B 34, Extension of proliferating epithelium in process of being included in the superficial loss of tissue.

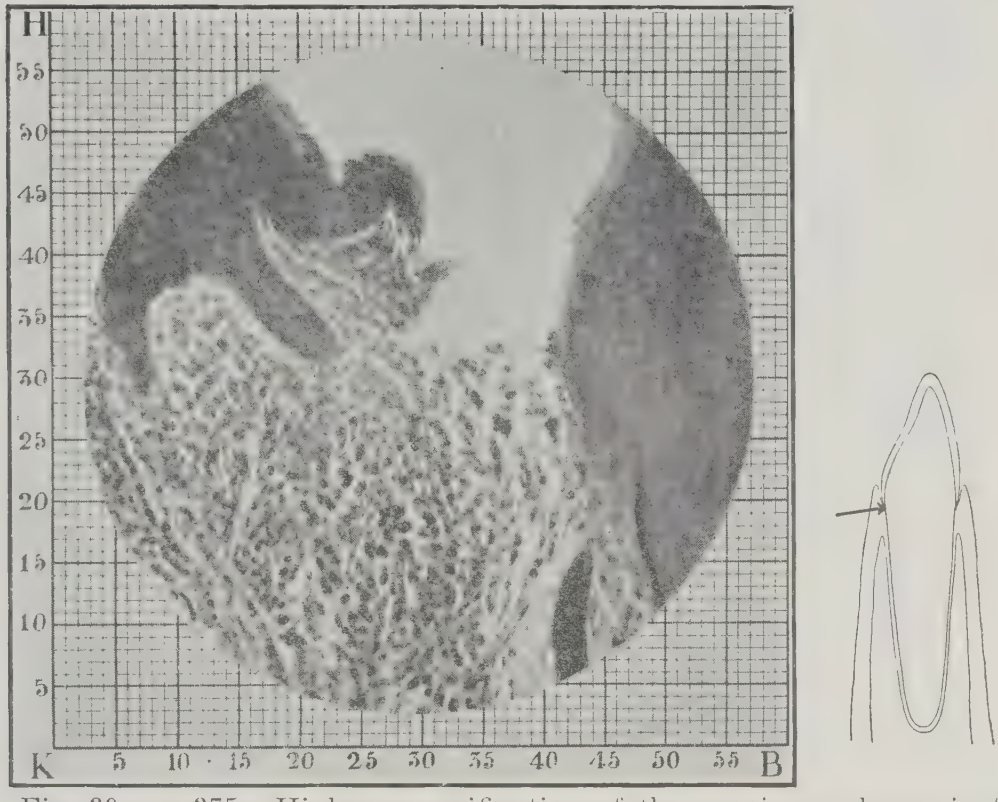


Fig. 39— x 375. Higher magnification of the specimen shown in the preceding illustration, showing ulceration of the connective tissue at the base of the gingival crevice.

H K 32 x K B 35, Ulceration at the base of the gingival crevice.
H K 14 x K B 43, Proliferating epithelial extension.
H K 50 x K B 20, Crevicular epithelium.

CHRONIC ULCERATIVE.

Due to increased virulence of the bacteria, or lowered resistance by the action of factors which may cause a disturbance of the capillary circulation, exacerbations of acute inflammation may occur. These frequently lead to an ulceration at the base of the gingival crevice and which may persist for long periods of time. They thus become chronic in character although having had their inception in an acute inflammatory process. However there is a tendency at all times, due to the natural resistance of the organism, for this process to merge into a chronic non-ulcerative condition (figs. 38 and 39).

PART III: RAREFYING PERICEMENTITIS FIBROSA.

This is a chronic and progressive lesion of the pericementum which results in the rarefaction and destruction of alveolar bone. The process consists of the formation, in the pericementum, of a new tissue which, on the one hand, changes the normal arrangement of the pericemental structures, and, on the other, replaces the bone substance of the alveolar process. These changes in the pericementum and the alveolar bone are concomitant lesions. This new tissue subsequently assumes the form of a fibrillar connective tissue (Figs. 40, 41, 42, 43, 44).

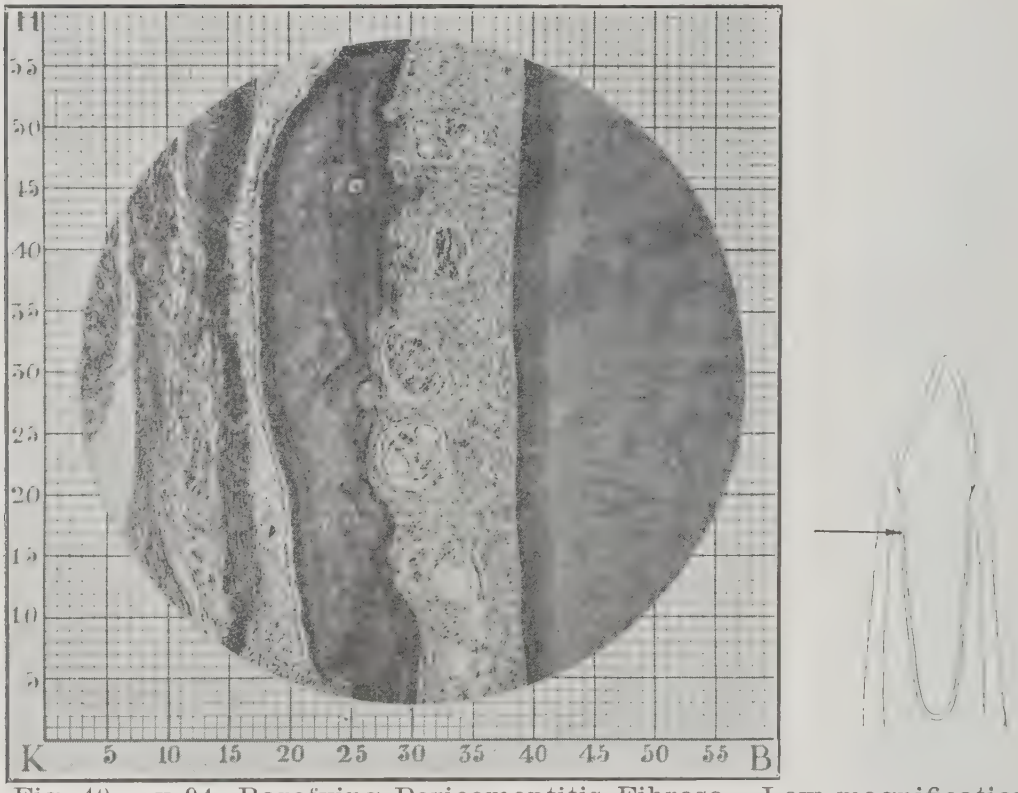


Fig. 40— x 94. Rarefying Pericementitis Fibrosa. Low magnification of tooth, pericementum and alveolar process, showing the formation of fairly large nodular masses of new tissue in the pericementum with resultant concavities in the inner alveolar wall.

H K 35 x K B 25, Alveolar process. H K 35 x K B 35, Pericementum. H K 35 x K B 40, Cementum. H K 35 x K B 45, Dentine.

H K 49 x K B 33, H K 39 x K B 33, H K 30 x K B 30, H K 28 x K B 30, H K 13 x K B 32, H K 5 x K B 35; Six nodular masses of new tissue in the pericementum.

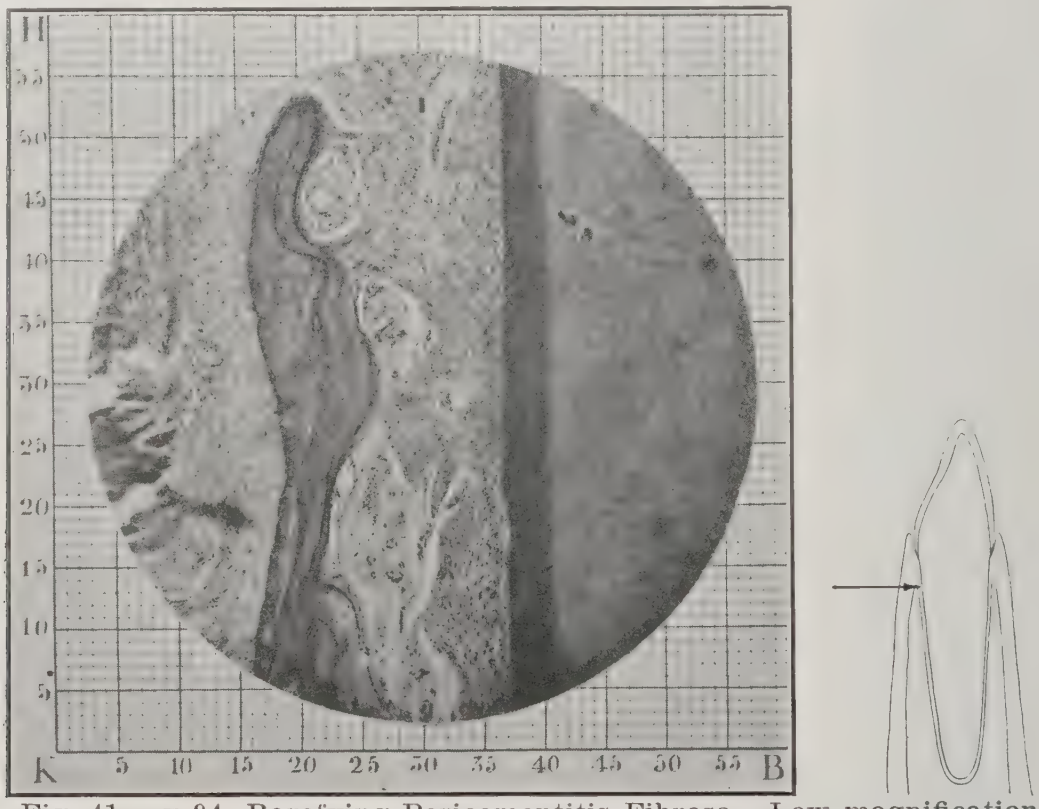
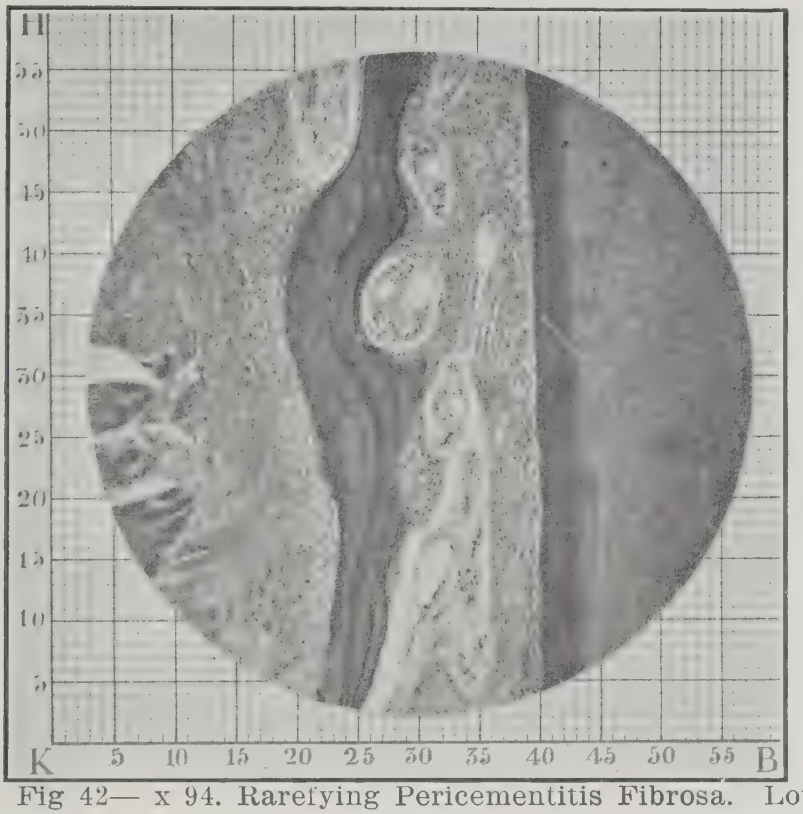


Fig. 41— x 94. Rarefying Pericementitis Fibrosa. Low magnification of tooth, pericementum and alveolar process showing nodular and irregular masses of new tissue in the pericementum.

H K 53 x K B 20, Crest of alveolar process. H K 45 x K B 30, Pericementum.
H K 45 x K B 23
H K 35 x K B 27 Nodular masses of new tissue in the pericementum.
H K 15 x K B 25, Large irregular mass of new tissue.
H K 55 x K B 40, Dentinal-cemental junction.



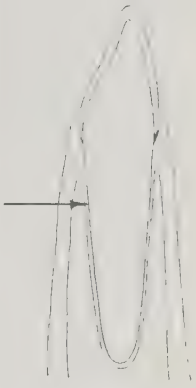


Fig 42— x 94. Rarefying Pericementitis Fibrosa. Low magnification of alveolar process, pericementum and tooth showing nodular and irregular masses of new tissue in the pericementum.

H K 40 x K B 24, Alveolar process. H K 50 x K B 37, Pericementum. H K 55 x K B 39, Border of cementum.

H K 45 x K B 31 }
H K 35 x K B 28 } Nodular masses of new tissue.

H K 13 x K B 34, Large irregular mass of new tissue.

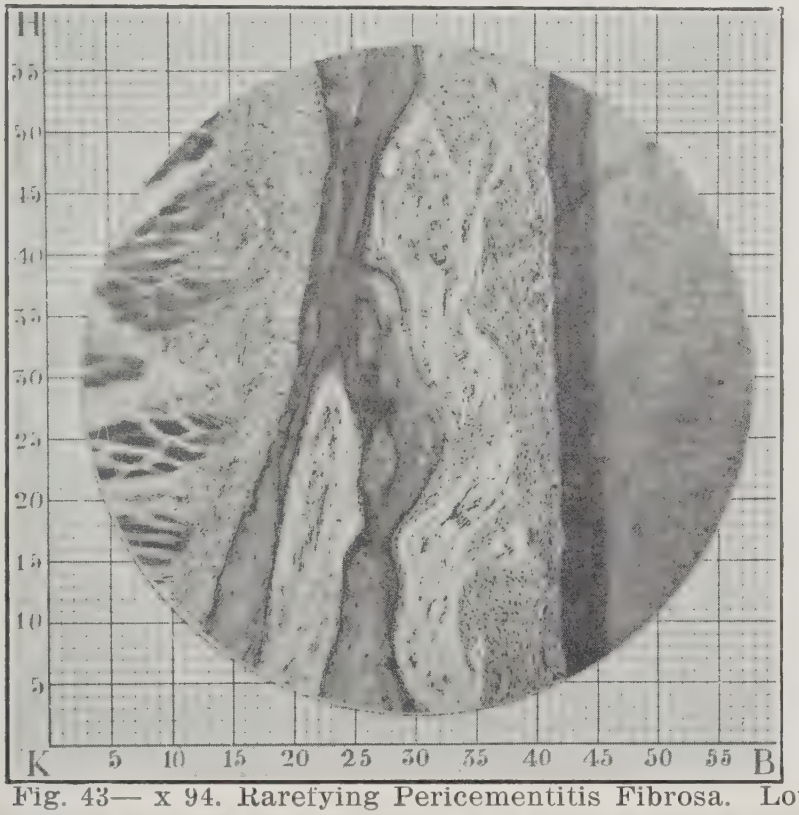


Fig. 43— x 94. Rarefying Pericementitis Fibrosa. Low magnification of alveolar process, pericementum and tooth showing two large irregular masses of new tissue in the pericementum, and one in the alveolar bone.

H K 52 x K B 25, Alveolar process. H K 53 x K B 35, Pericementum.
H K 55 x K B 41, Cemental border.

H K 43 x K B 30 }
H K 15 x K B 32 } Large irregular masses of new tissue in pericementum.

H K 20 x K B 23, Large mass of new tissue in alveolar process.

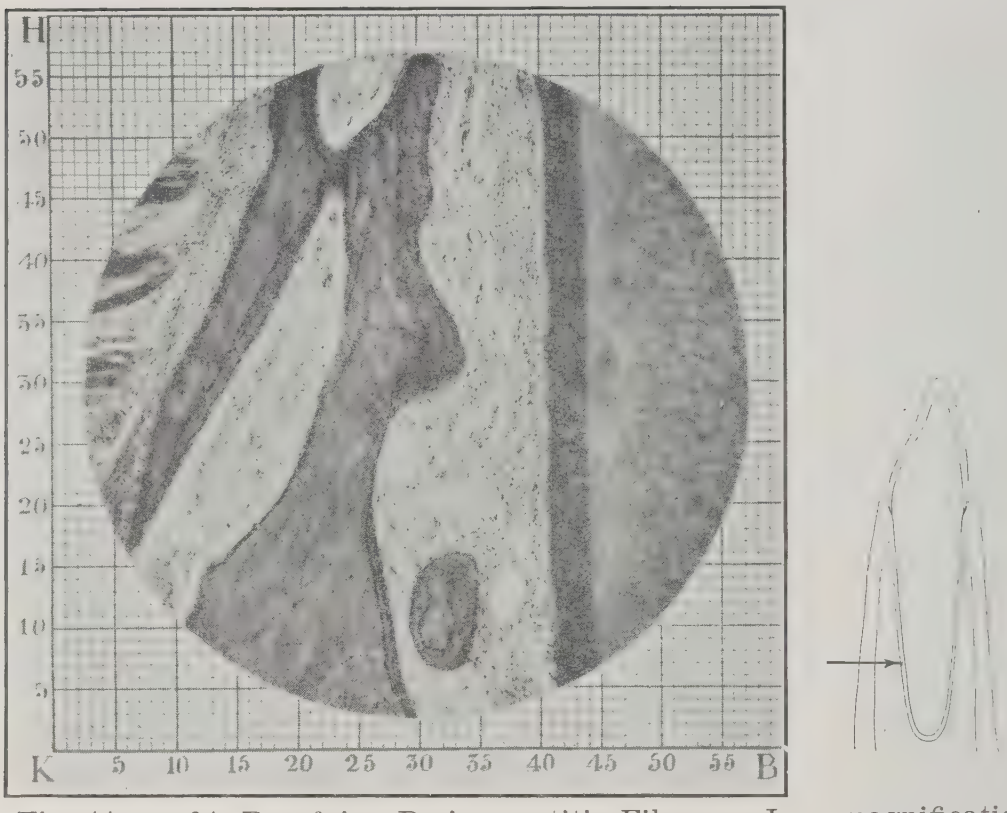


Fig. 44— x 94. Rarefying Pericementitis Fibrosa. Low magnification of alveolar process, pericementum and tooth showing masses of new tissue.

H K 33 x K B 27, Alveolar process.
H K 30 x K B 33, Pericementum.
H K 25 x K B 42, Cementum.
H K 40 x K B 33 } Masses of new tissue in pericementum.
H K 20 x K B 32 }
H K 25 x K B 17, Mass of new tissue in alveolar process.
H K 10 x K B 32, Segment of alveolar bone.

In this new tissue, the reaction around the blood-vessels is a conspicuous feature. There is a proliferative change of the endothelial cells lining the blood-vessels, leading, in many instances, to partial or complete occlusion of the vessel lumen. Endothelial cells are to be seen, also, in the new tissue surrounding the blood-vessels, accompanied by occasional lymphocytes and plasma cells. A few fibroblasts are present. In other words, there is manifested a chronic and progressive tissue response wherein new fibroblasts make their appearance during the progress of a very low grade of chronic inflammation (Figs. 45, 46, 47, 48, 49).

In the production of this reaction, however, there is no significant development of new capillaries, and the tissue cannot be looked upon as typical granulation tissue. Nevertheless, there has taken place a new-growth of tissue, its various elements constituting at times a diffuse new structure, and, at other times, nodular and irregular masses of new tissue growth (Figs. 50, 51, 52, 53, 54, 55, 56, 57, 58, 59).

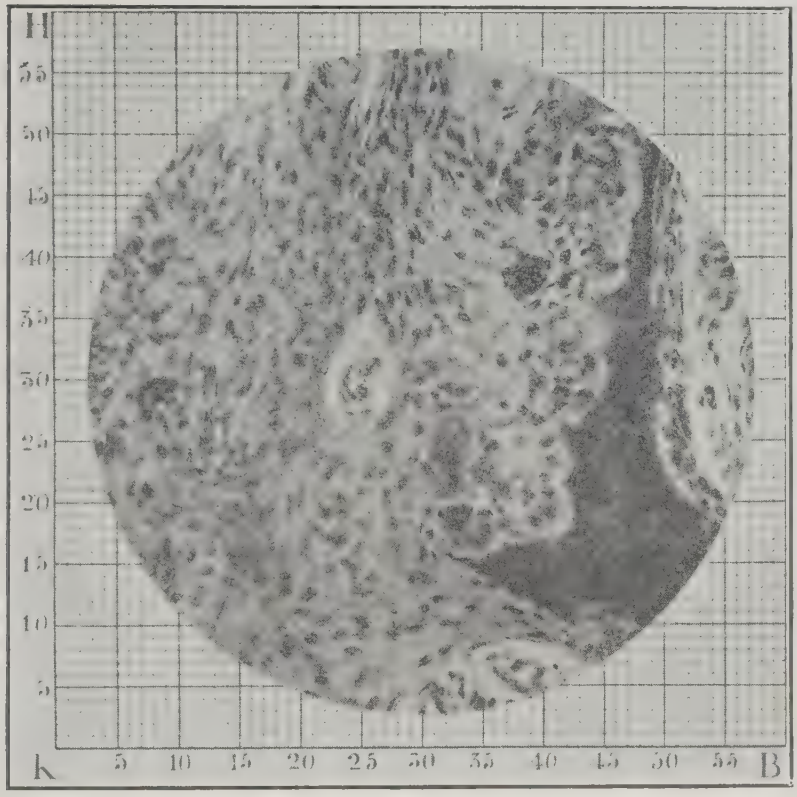


Fig. 45— x 375. Rarefying Pericementitis Fibrosa. Small nodule of new tissue in the body of the pericementum.

H K 30 x K B 25, Small nodular mass of new tissue.
H K 15 x K B 45, Small segment of alveolar bone undergoing absorption.
H K 22 x K B 35 Osteoclast.
H K 30 x K B 25, Blood-vessel in small mass of new tissue.

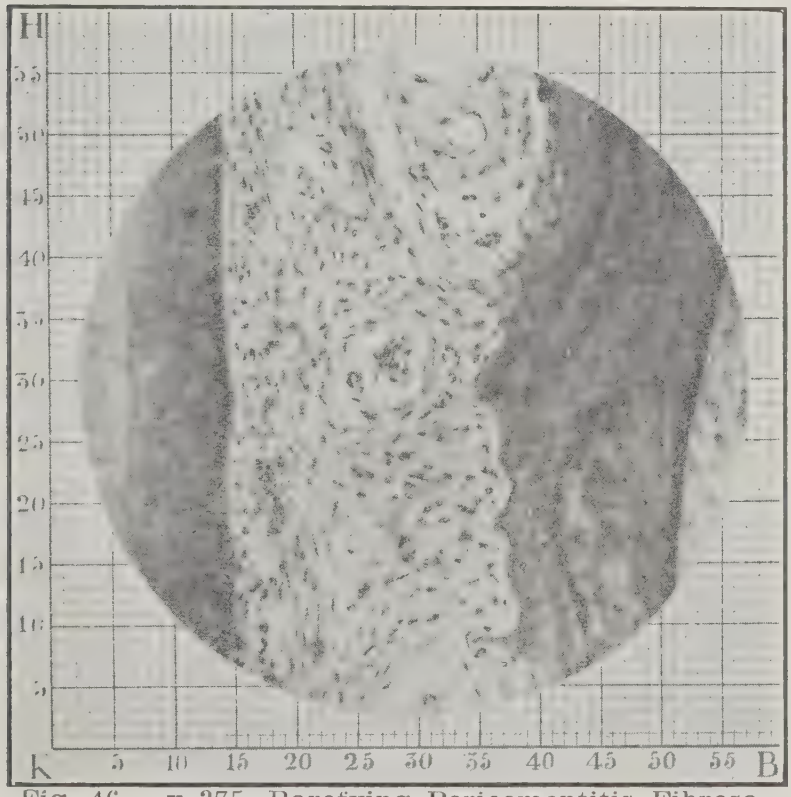
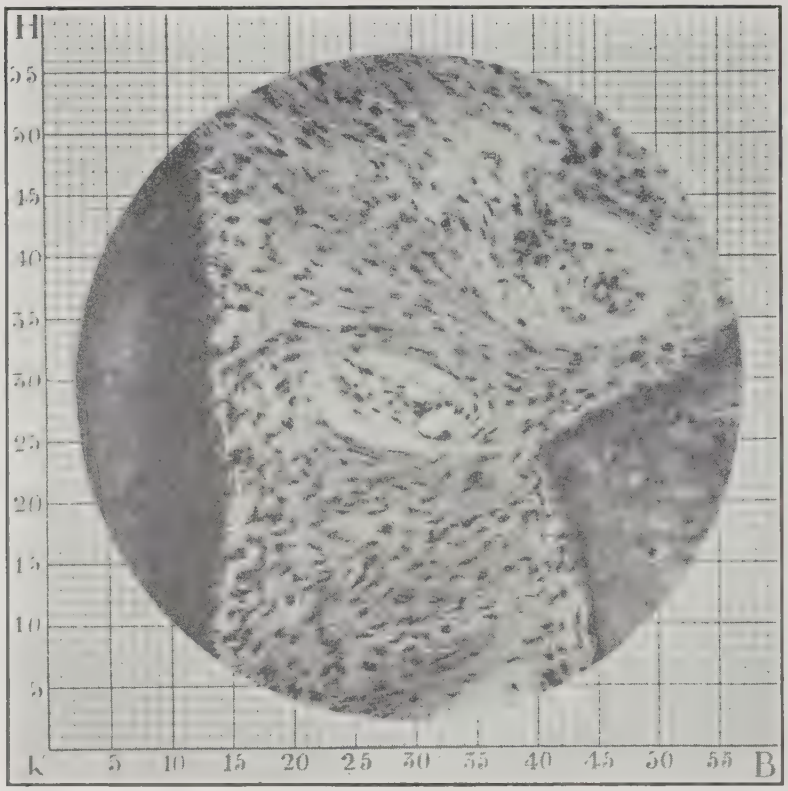


Fig. 46— x 375. Rarefying Pericementitis Fibrosa. Small nodule of new tissue in the body of the pericementum.

H K 31 x K B 27, Small nodule of new tissue in the pericementum.
H K 43 x K B 6, Dentinal-cemental junction.
H K 53 x K B 14, Border of the cementum.
H K 25 x K B 37, Inner surface of alveolar bone. Note its eroded appearance.
H K 32 x K B 27. Blood-vessel in small nodule.



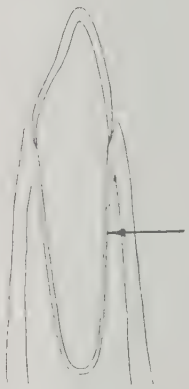
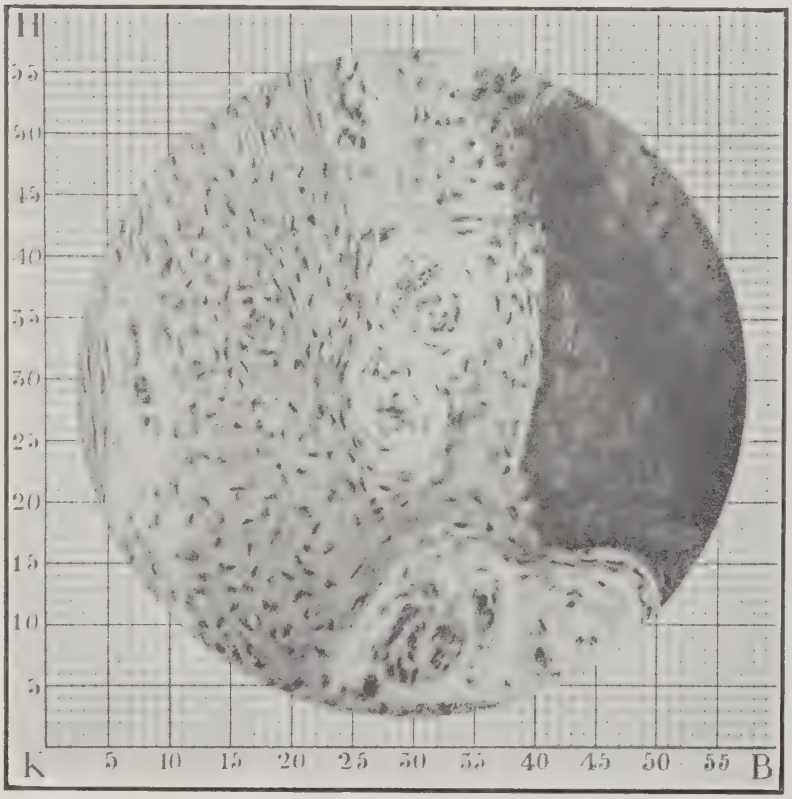


Fig. 47— x 375. Rarefying Pericementitis Fibrosa. Small nodule of new tissue in the pericementum.

H K 28 x K B 27, Small nodule of new tissue.
H K 27 x K B 31, Blood-vessel in nodule.
H K 37 x K B 42, Large mass of new tissue.
H K 20 x K B 14, Cemental border.
H K 20 x K B 41, Alveolar wall.
H K 15 x K B 30, Principal fibres of the pericementum.



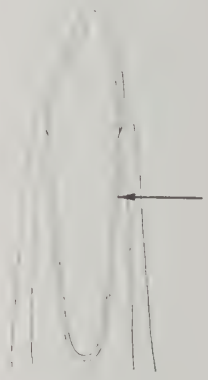


Fig. 48— x 375. Rarefying Pericementitis Fibrosa. Two small nodules of new tissue in the pericementum.

H K 36 x K B 31, Upper nodule of new tissue.
H K 27 x K B 28, Lower nodule of new tissue.
H K 32 x K B 28, Blood-vessel in lower nodule.
H K 23 x K B 38, Alveolar wall.

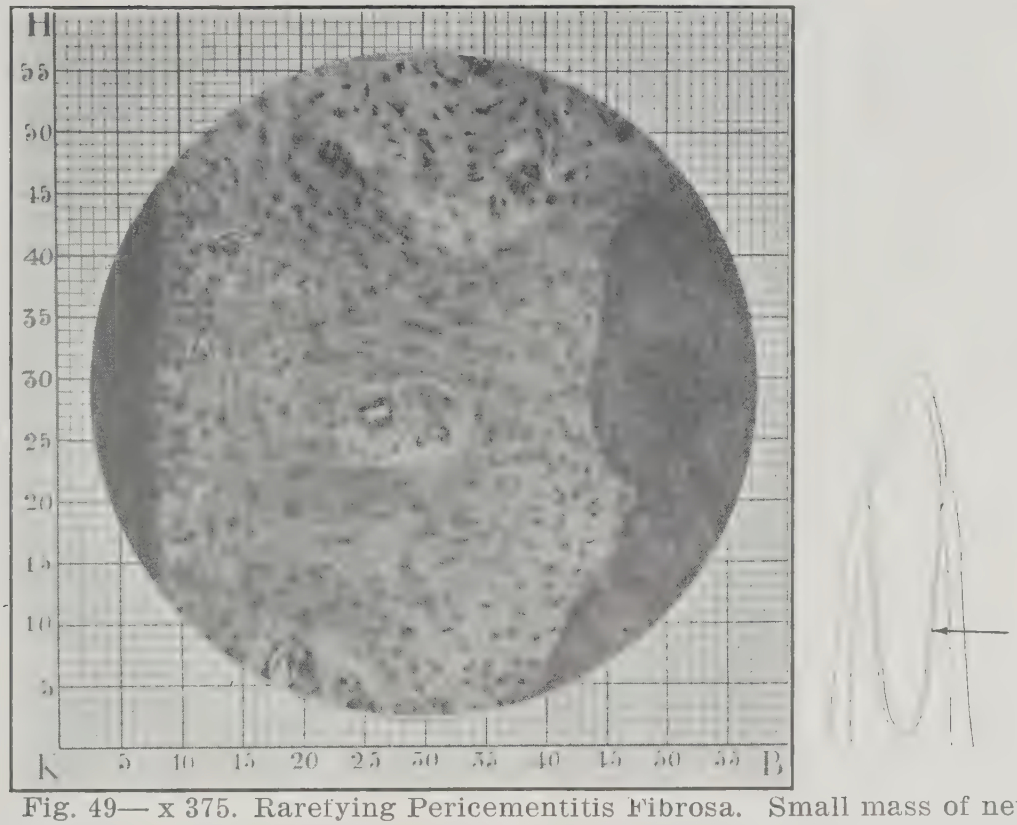


Fig. 49— x 375. Rarefying Pericementitis Fibrosa. Small mass of new tissue in the body of the pericementum.

H K 27 x K B 30, Small nodule of new tissue. H K 27 x K B 25, Blood-vessel in nodular mass. H K 32 x K B 28, H K 20 x K B 25, Principal fibres of the pericementum. H K 45 x K B 8, Cemental border. H K 25 x K B 43, Alveolar wall.

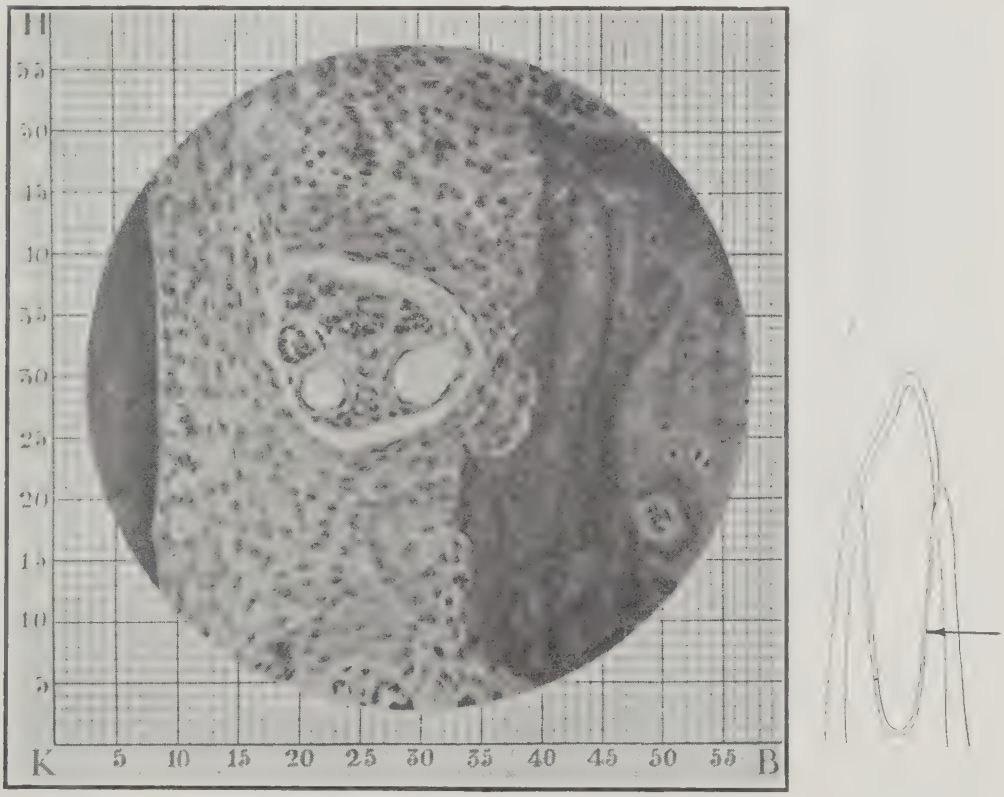


Fig. 50— x 375. Rarefying Pericementitis Fibrosa. Large mass of new tissue encroaching upon the surface of the alveolar process with resultant absorption of bone. Note the tiny bay-like excavation opposite the mass of new tissue.

H K 33 x K B 25, Large nodular mass of new tissue. H K 36 x K B 20, H K 32 x K B 20, Blood-vessels in new tissue. H K 25 x K B 38, Excavation of the alveolar wall. Note the row of osteoblasts on the border of the excavation. H K 45 x K B 8, Cemental border. H K 40 x K B 31, Principal fibres of pericementum. H K 15 x K B 34, Surface of alveolar bone.

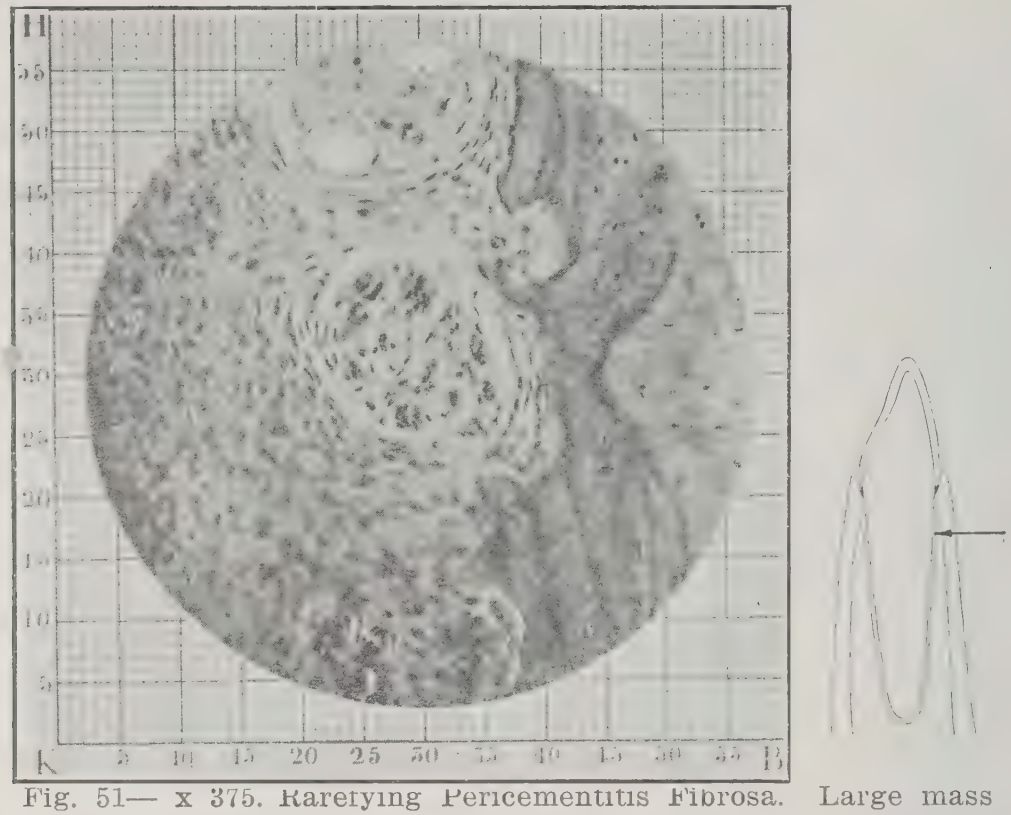


Fig. 51— x 375. Rarefying Pericementitis Fibrosa. Large mass of new tissue encroaching upon the surface of the alveolar process with resultant beginning of bone absorption. Note small inlet and large excavation in the alveolar wall.

H K 30 x K B 28, Large nodule of new tissue. H K 26 x K B 28, Blood-vessel in new tissue. H K 40 x K B 41, Small inlet in alveolar wall. H K 24 x K B 29, Large excavation. H K 20 x K B 28, Principal fibres of pericementum.

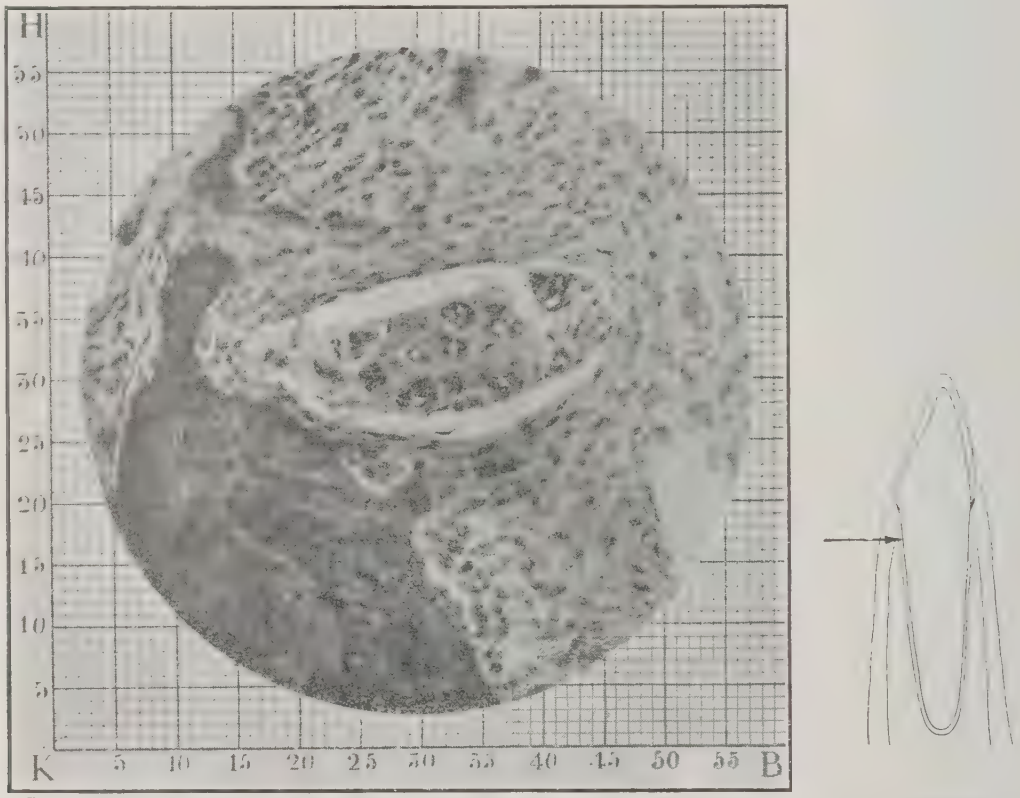


Fig. 52— x 375. Rarefying Pericementitis Fibrosa. Large mass of new tissue encroaching upon the crest of the alveolar process with the resultant absorption of a large portion of the crest. Note the tiny inlet below the mass of new tissue.

H K 40 x K B 12, Tip of the alveolar crest. H K 32 x K B 32, Large mass of new tissue. Note the numerous blood-vessels. H K 30 x K B 12, Base of excavation in alveolar bone. H K 22 x K B 26, Tiny inlet in alveolar surface.

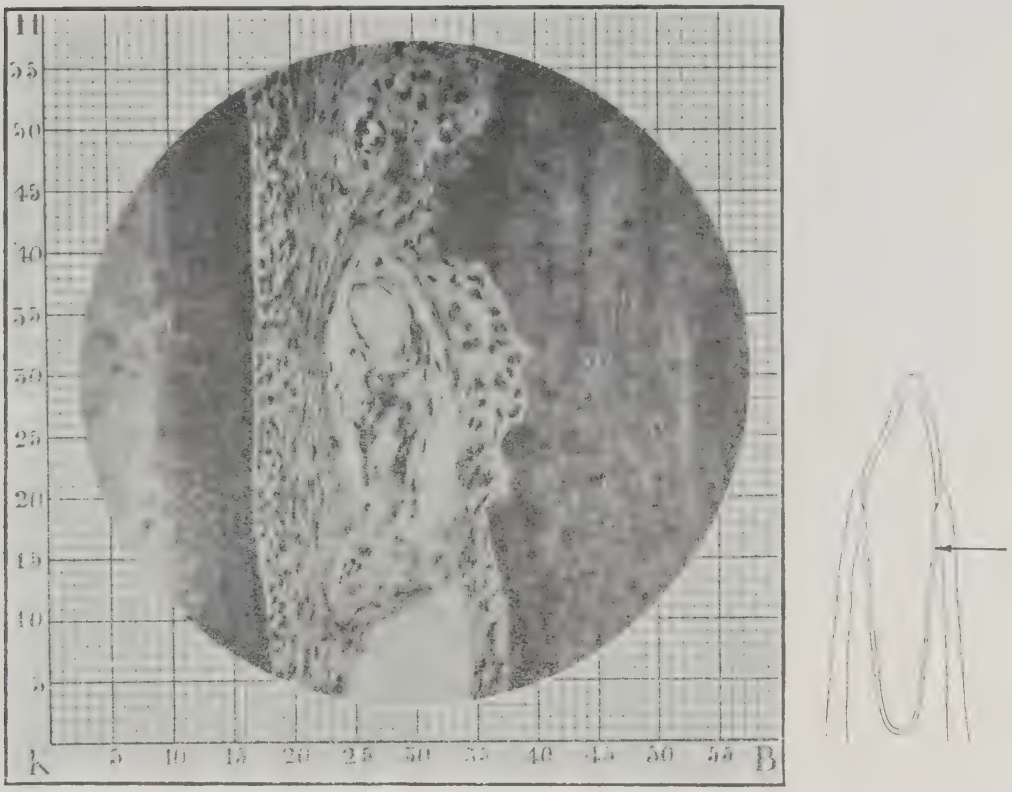


Fig. 53— x 375. Rarefying Pericementitis Fibrosa. Large mass of new tissue near the surface of the alveolar wall with typical excavation in the bone. Note the row of osteoblasts on the excavation border.

H K 25 x K B 30. Large mass of new tissue. H K 31 x K B 39. Base of excavation.

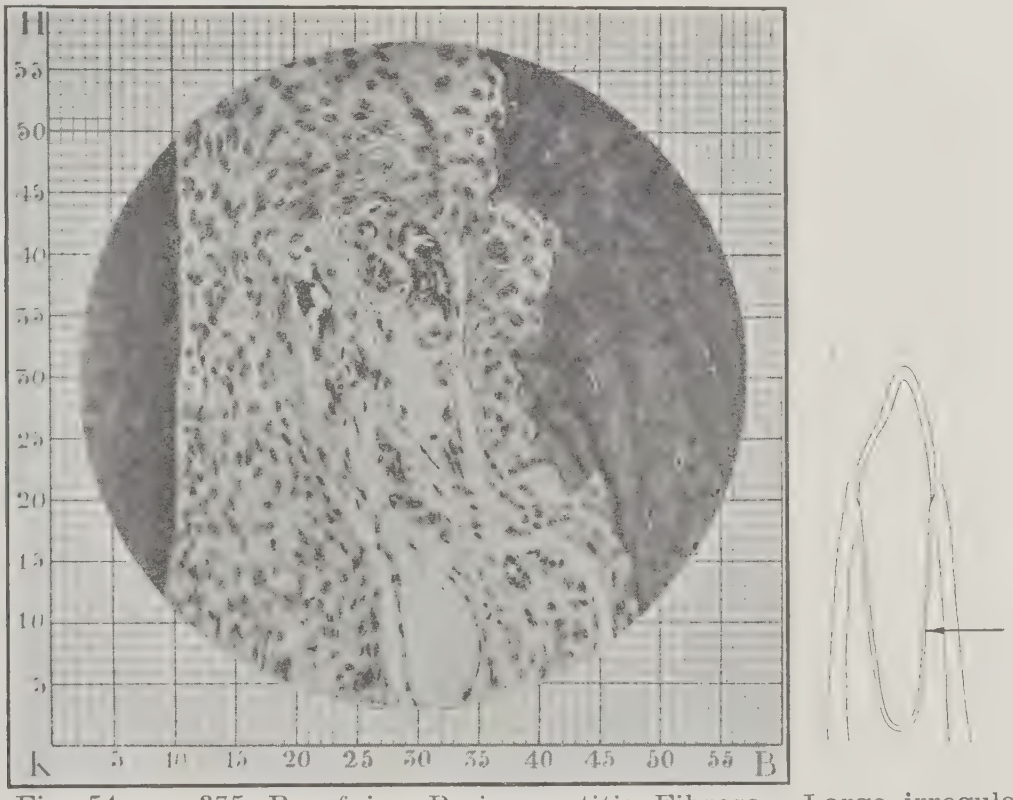
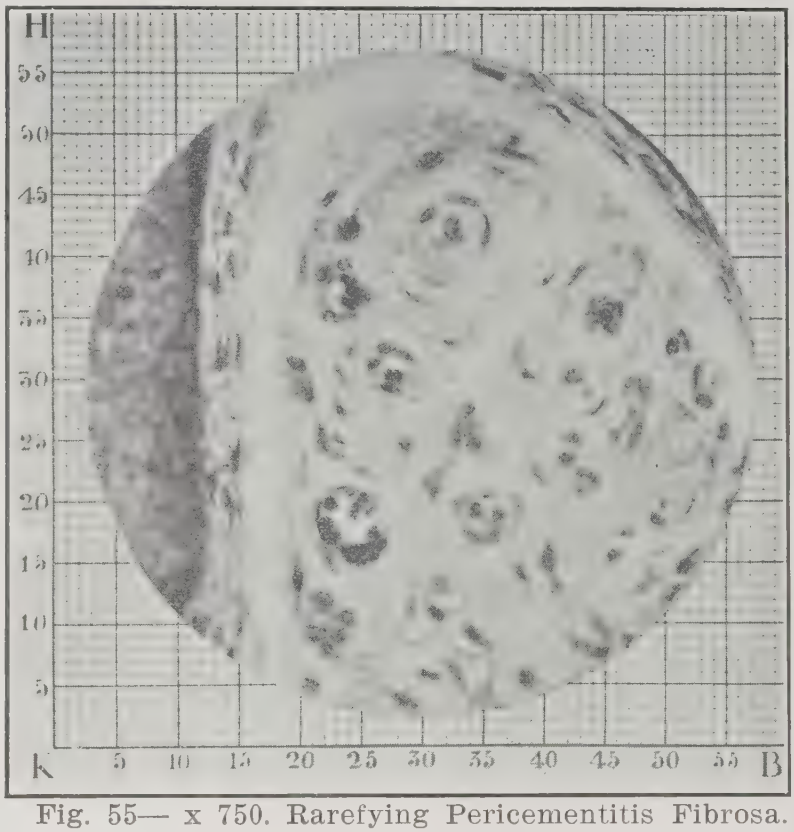


Fig. 54— x 375. Rarefying Pericementitis Fibrosa. Large irregular mass of new tissue near the surface of the alveolar bone. Note the eroded appearance of the bone surface.

H K 44 x K B 27, Upper margin of irregular mass of new tissue.
H K 4 x K B 28 } Lower borders of mass of new tissue.
H K 9 x K B 46 }
H K 38 x K B 31 Blood-vessels in new tissue.
H K 14 x K B 38
H K 40 x K B 40, Base of excavation of bony surface.
H K 49 x K B 10, Cemental border.



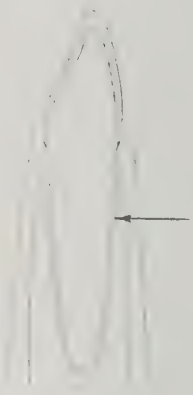
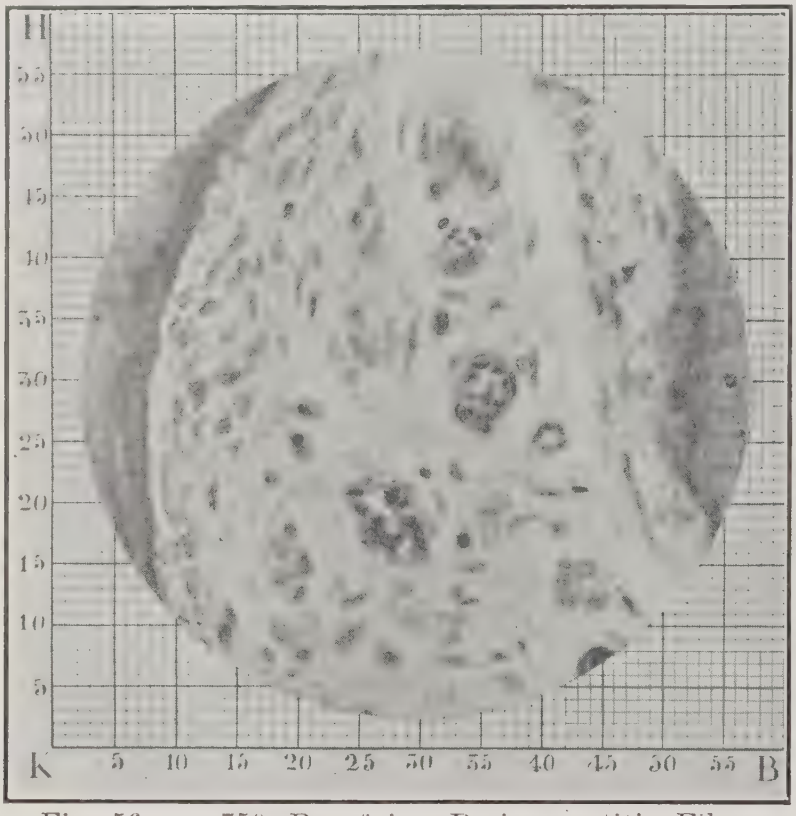


Fig. 55— x 750. Rarefying Pericementitis Fibrosa. High magnification of new tissue. Note proliferation of endothelial cells lining blood-vessels; also types of cells in new tissue surrounding the blood-vessels.

H K 43 x K B 33, Complete occlusion of blood-vessel lumen. H K 19 x K B 35, Partial occlusion of blood-vessel lumen. H K 30 x K B 20, Endothelial cells in the new tissue. H K 42 x K B 24, Plasma cell. H K 39 x K B 43, H K 37 x K B 30: Fibroblasts. H K 18 x K B 25, Large blood-vessel.



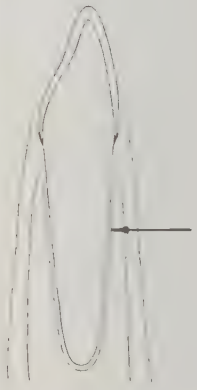


Fig. 56— x 750. Rarefying Pericementitis Fibrosa. High magnification of new tissue.

H K 48 x K B 33, Complete occlusion of blood-vessel lumen through proliferation of the endothelial cells. H K 40 x K B 34, Partial occlusion of blood-vessel lumen. H K 18 x K B 24, H K 12 x K B 25: Endothelial cells in new tissue. H K 28 x K B 20, H K 25 x K B 20, H K 17 x K B 34: Plasma cells. H K 23 x K B 30, Lymphocyte. H K 41 x K B 39, Fibroblast. H K 30 x K B 35, Large blood-vessel.

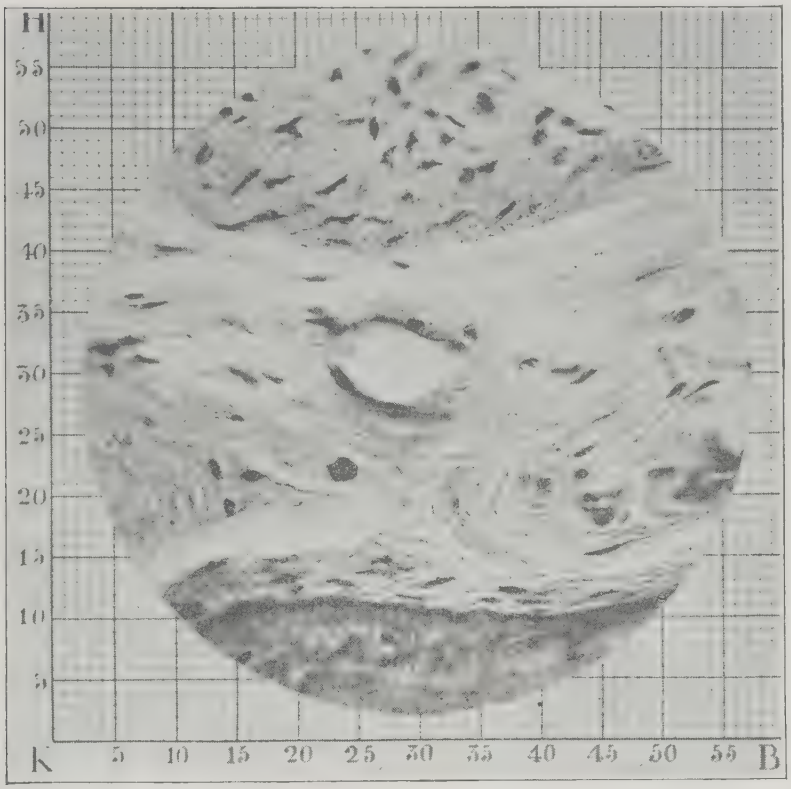


Fig. 57— x 750. Rarefying Pericementitis Fibrosa. High magnification of new tissue.

H K 23 x K B 23, Plasma cell in new tissue.

H K 21 x K B 40 } Lymphocytes in new tissue.
H K 22 x K B 43 }

H K 29 x K B 53 } Fibroblasts in new tissue.
H K 16 x K B 45 }

H K 40 x K B 30, Margin of mass of new tissue. H K 45 x K B 30, Normal pericementum. H K 10 x K B 36, Surface of alveolar bone.

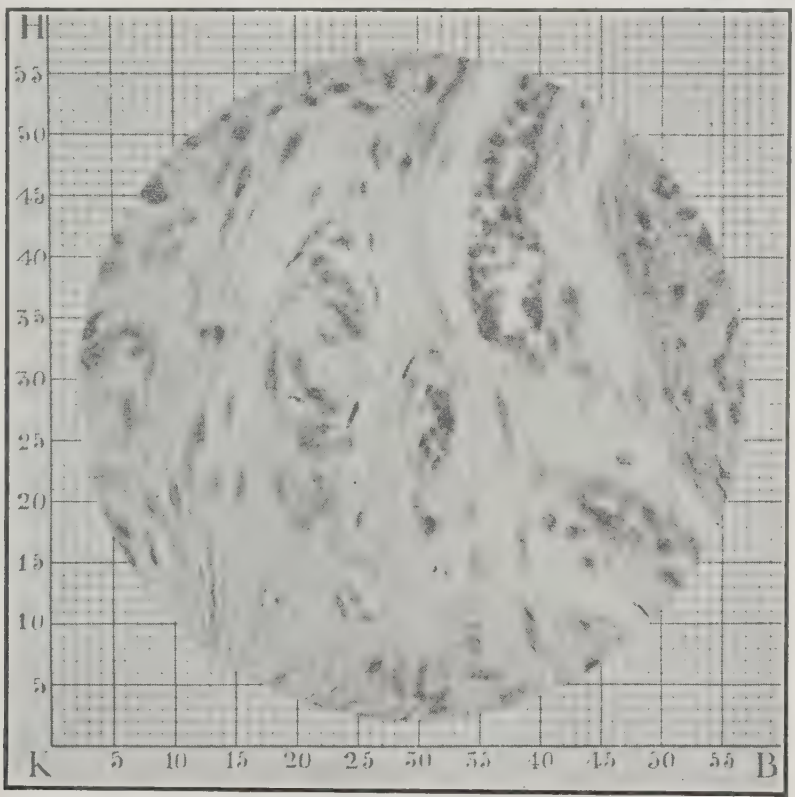


Fig 58— x 750. Rarefying Pericementitis Fibrosa. High magnification of new tissue.

H K 35 x K B 35, Large blood-vessel.

H K 20 x K B 45, Partial occlusion of blood-vessel lumen. Note the endothelial cells in the new tissue surrounding the same blood-vessel.

H K 35 x K B 24, Endothelial cells in the new tissue.

H K 31 x K B 29 } Fibroblasts.
H K 27 x K B 25 }

H K 48 x K B 29, Lymphocyte.

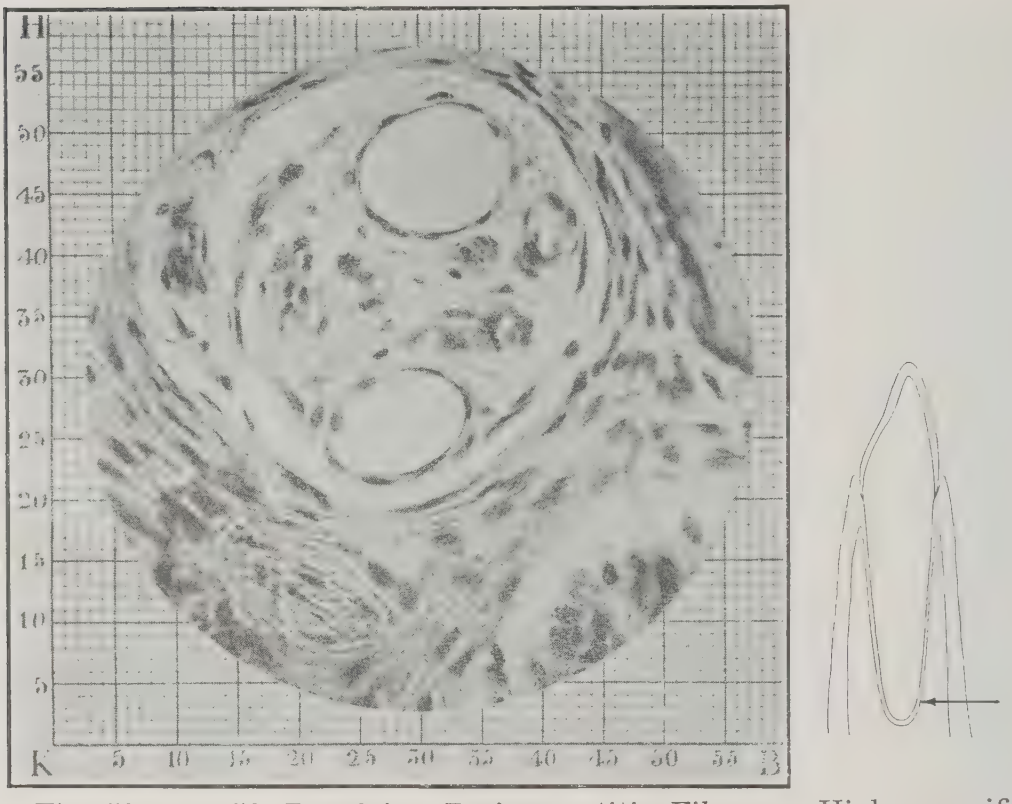


Fig. 59— x 750. Rarefying Pericementitis Fibrosa. High magnification of nodular mass of new tissue.

H K 34 x K B 36, Partial occlusion of blood-vessel lumen.
H K 38 x K B 11, Complete occlusion of blood-vessel lumen.
H K 39 x K B 24, Plasma cell.
H K 53 x K B 32, Fibroblast.
H K 32 x K B 27, Lymphocyte.
H K 33 x K B 33, Endothelial cell in new tissue.
H K 15 x K B 25, Normal pericemental tissue.

The rarefaction of the alveolar process is the result of involvement, by the new tissue, of the osteogenetic tissue on the exterior of the bone, and the setting up of the mechanism of bone absorption. These areas are manifested by great cell activity. However, other areas of bone loss seem to be associated with nutritional disturbances caused by this new tissue, and atrophic changes follow in the bone. Starting with a fairly simple process, the final rarefaction of the bone is a complicated melting-down process.

As stated previously, the reaction around the blood-vessels, characterized by the presence of endothelial cells, along with occasional lymphocytes, plasma cells, and fibroblasts, is the prominent feature of this new tissue. Through increase in size of masses of this tissue lying in the body of the pericementum, or through proximity to the surface of the alveolar bone, of the blood-vessels manifesting the reaction, these same cells encroach upon the bony surface. Accompanied by osteoblasts and osteoclasts, the absorption of bone is seen to take place, with the formation of little bay-like depressions into the surface of the alveolar wall. Into these inlets, the new tissue extends in finger-like projections along the blood-vessels, carrying inwards reactions similar to those seen upon the surface, and producing within the bone an internal rarefaction (Figs. 60, 61).

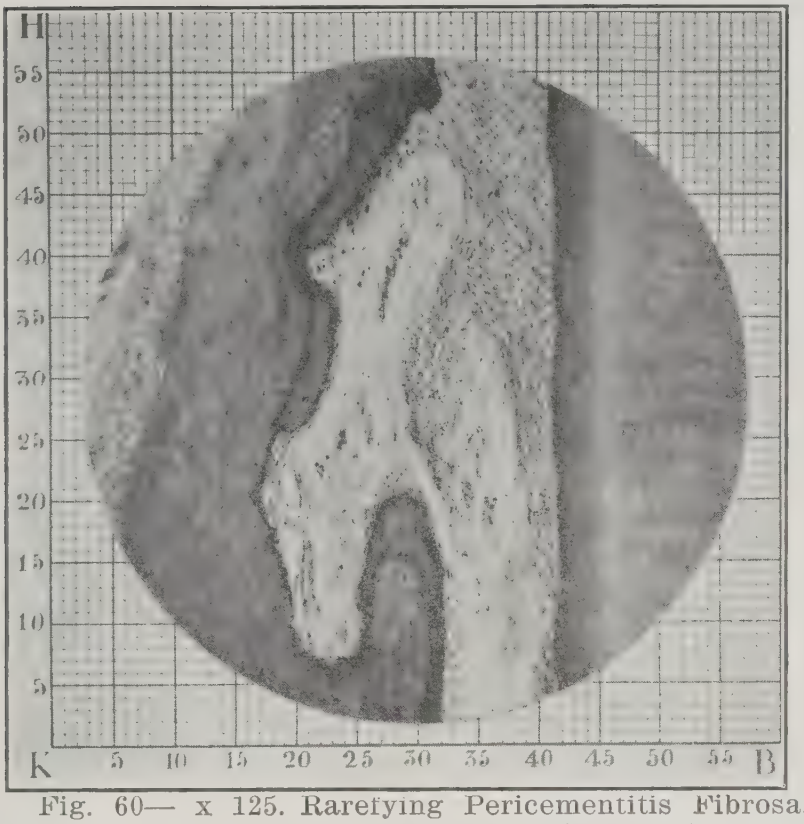


Fig. 60— x 125. Rarefying Pericementitis Fibrosa. Alveolar rarefaction. Large bay-like depression in the alveolar wall. Note that it is filled with new tissue, an extension from the pericementum.

H K 30 x K B 15, Alveolar process. H K 7 x K B 23, Extremity of bay-like depression. H K 30 x K B 25, Large mass of new tissue. H K 50 x K B 38, Normal pericementum, H K 10 x K B 35, New tissue in pericementum. H K 54 x K B 41, Cemental border. H K 30 x K B 10, External surface of alveolar process.

Fig. 61— x 125. Rarefying Pericementitis Fibrosa. Alveolar rarefaction. Large bay-like depression in the alveolar bone filled with new tissue which has replaced the bone by extension from the pericementum.

H K 30 x K B 5, Normal pericementum. H K 40 x K B 30, Alveolar process. H K 45 x K B 42, Extremity of large bay-like cavity. H K 20 x K B 35, New tissue filling depression. H K 25 x K B 46, External surface of alveolar process.

The rarefying process is frequently characteried, in its early stages, by the appearance at certain intervals along the wall of the alveolar bone, of fairly regular indentations formed by the encroachment of small nodular masses of new tissue (Figs. 62, 63, 64, 65).

Another typical feature is the appearance on the surface of the bone of irregular excavations, at the border of which is arranged a row of osteoblasts, each cell resting in a small concavity. In these larger excavations, masses of new tissue are to be seen (Figs. 66, 67).

Another characteristic of certain histological pictures is the presence of small transverse canals or channels with ragged walls, which frequently perforate the alveolar bone to a considerable distance from the surface. These small canals contain finger-like extensions of new tissue (Figs. 68, 69, 70, 71).

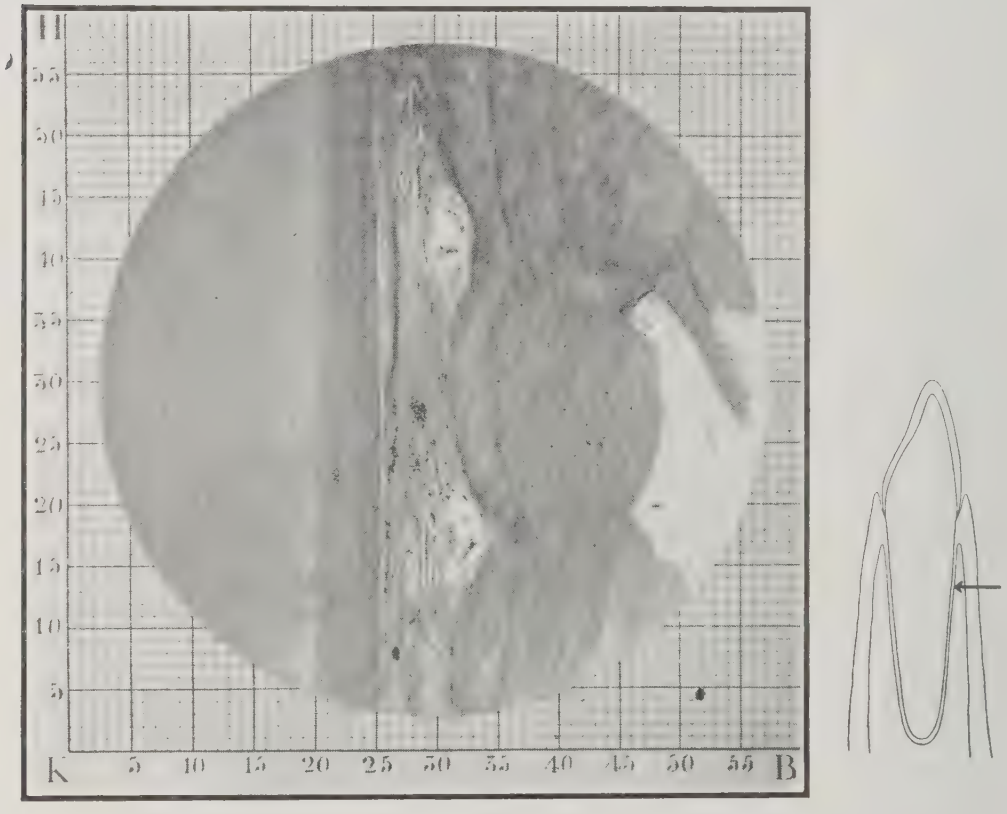


Fig. 62— x 94. Rarefying Pericementitis Fibrosa. Rarefaction of the alveolar process. Indentation.

H K 43 x K B 32
H K 18 x K B 33
} Indentations of the inner wall of the alveolar process.

H K 30 x K B 24, Cementum.

H K 30 x K B 28, Pericementum. Note that at this level the pericementum and cementum are of equal thickness.

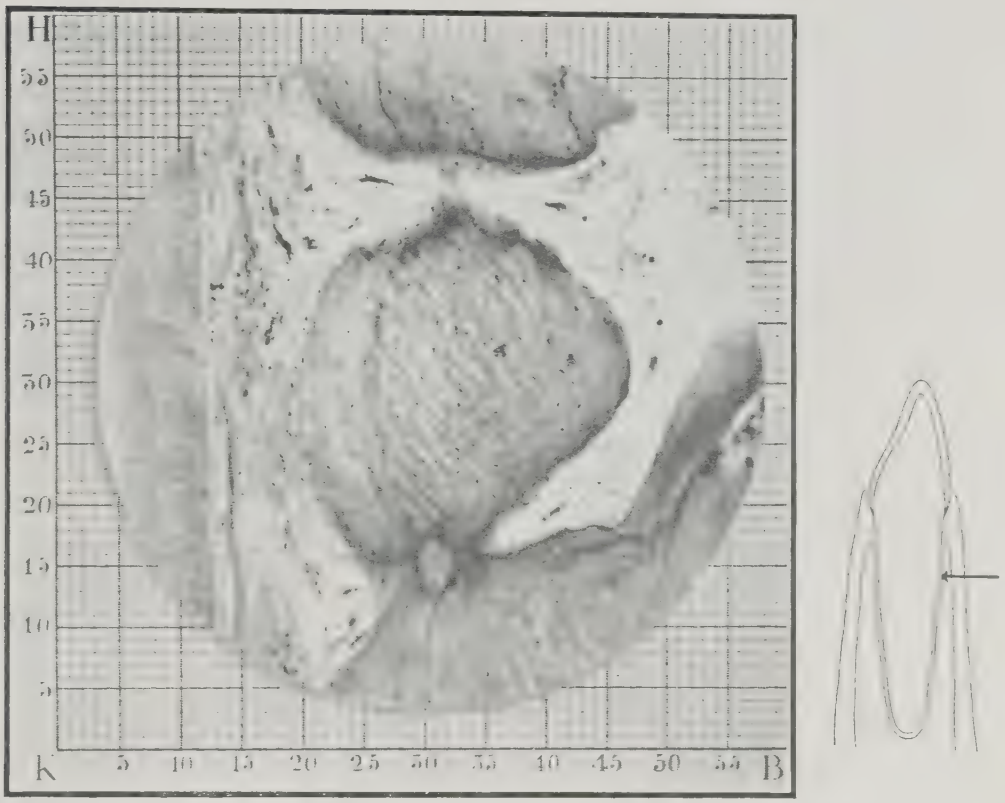


Fig. 63— x 125. Rarefying Pericementitis Fibrosa. Alveolar rarefaction. Indentation more advanced, leading to segmentation.

H K 33 x K B 35, Portion of alveolar bone in process of separation from adjacent bone. H K 14 x K B 27, H K 18 x K B 36: Indentation process tending to formation of continuous channel. H K 47 x K B 28, Indentation becoming a channel and thus producing segmentation of bone.

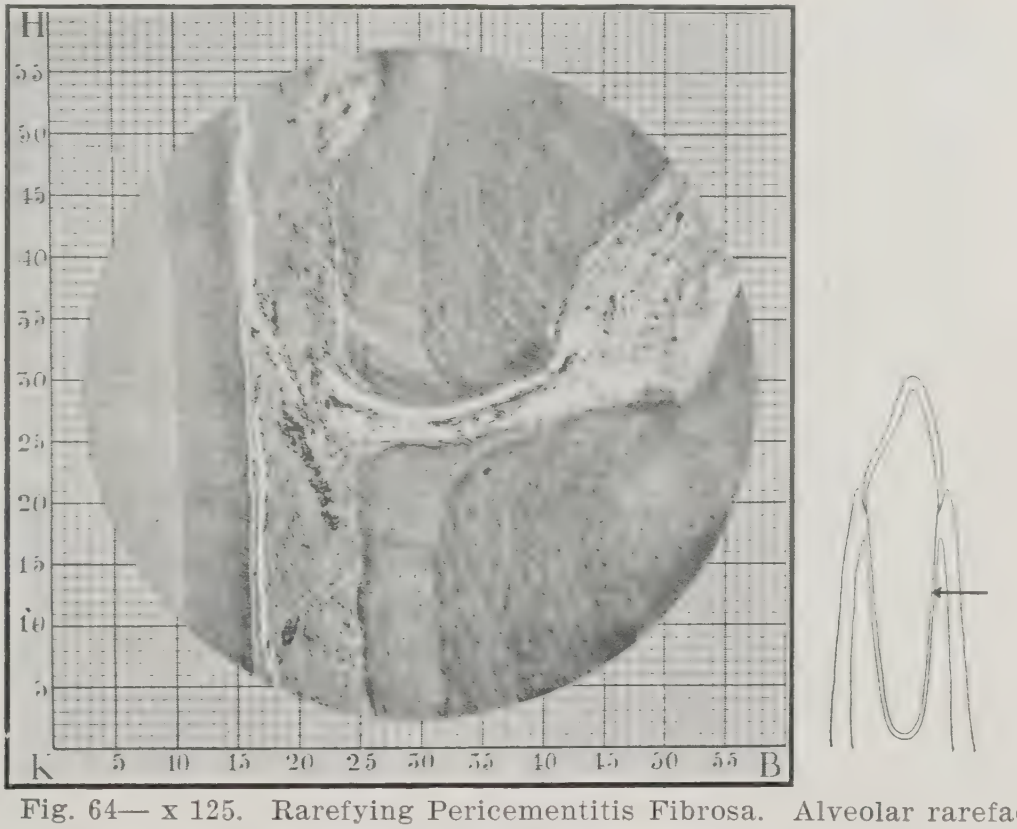


Fig. 64— x 125. Rarefying Pericementitis Fibrosa. Alveolar rarefaction. Channel produced as a result of the indentation process.

H K 54 x K B 24, Early indentation. H K 28 x K B 28, Channel formed through indentation process. H K 26 x K B 20, H K 34 x K B 18: Blood-vessels of pericementum much dilated. H K 15 x K B 14, Cementum. H K 15 x K B 22, Pericementum.

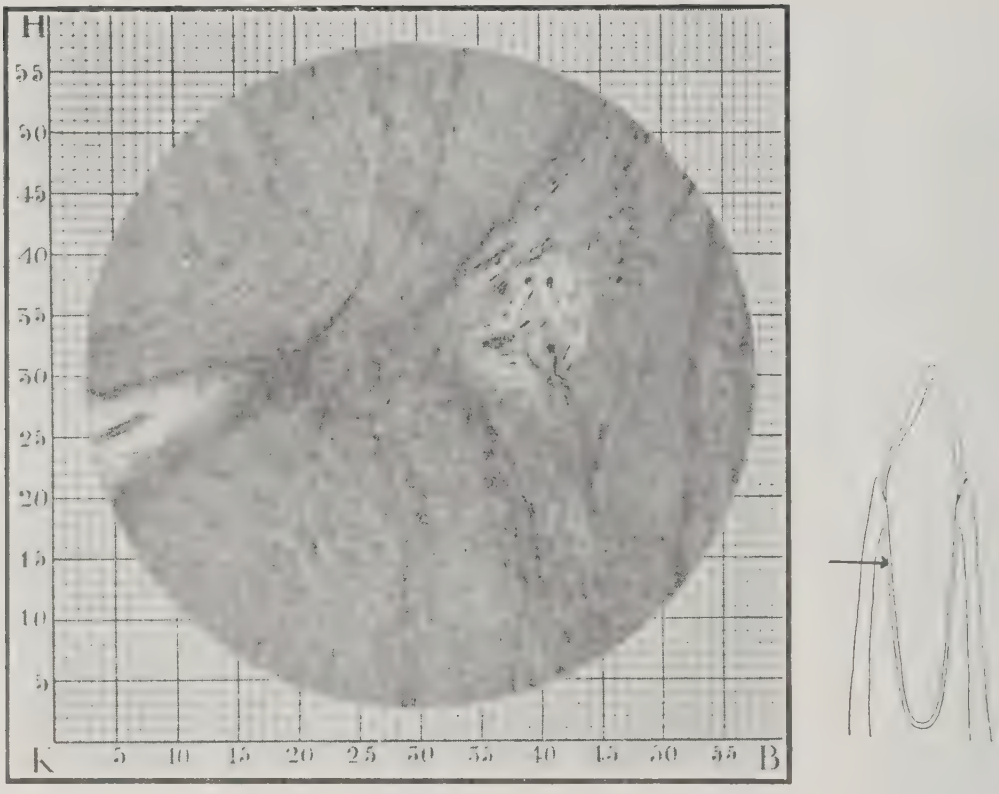


Fig. 65— x 375. Rarefying Pericementitis Fibrosa. Alveolar rarefaction. Indentation. Another example of the tendency of the indentation process to form channels through the alveolar bone.

H K 30 x K B 15 } Indentations.
H K 35 x K B 35 }

H K 30 x K B 52, Cemental surface. H K 30 x K B 48, Pericementum.

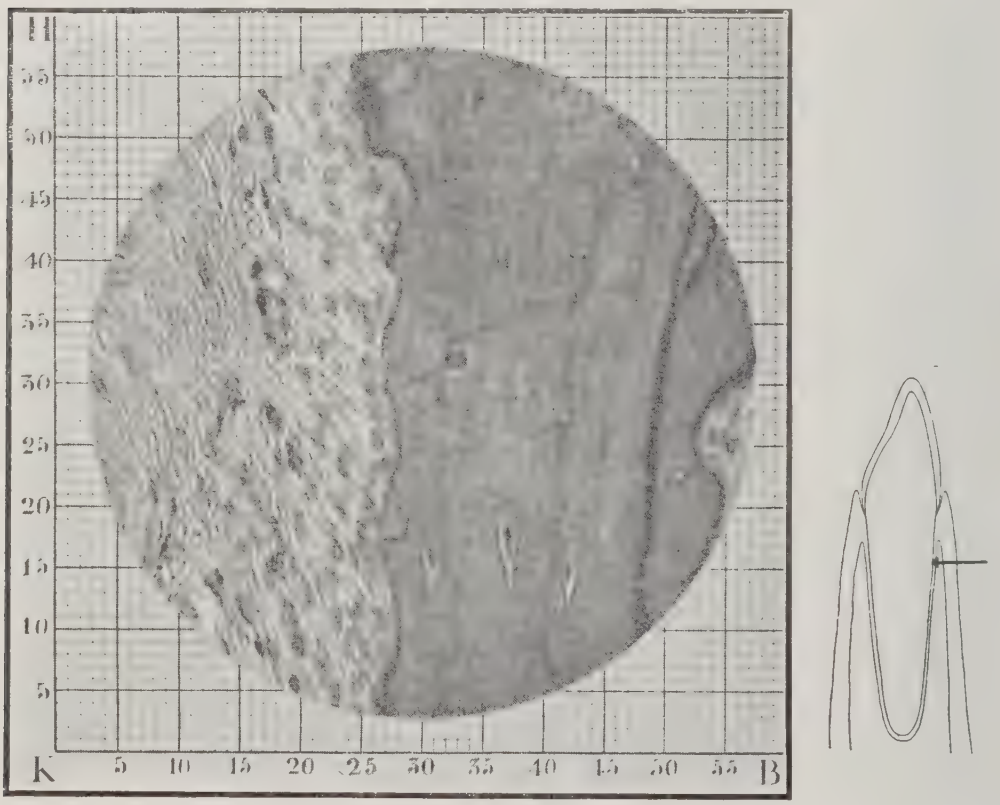


Fig. 66— x 500. Rarefying Pericementitis Fibrosa. Alveolar rarefaction. Excavation of the surface of the alveolar bone. A layer of osteoblasts is lying in close adaptation to the surface of the bone, which is undergoing absorption.

H K 49 x K B 25 to H K 18 x K B 27. This shows an irregular border, due to absorption. H K 30 x K B 25, H K 26 x K B 26: Osteoblasts. H K 18 x K B 37, Bone cell in lacuna.

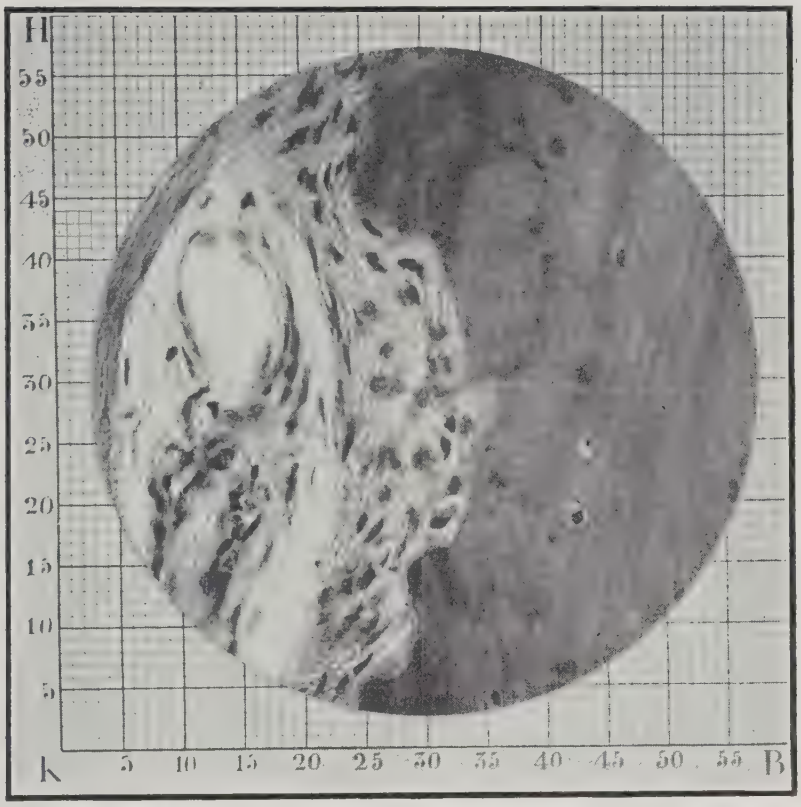


Fig. 67— x 500. Rarefying Pericementitis Fibrosa. Alveolar rarefaction. Excavation on the surface of the alveolar border.

H K 44 x K B 25 to H K 6 x K B 25, Large, irregular excavation. H K 19 x K B 31, Osteoblast. H K 35 x K B 14, Blood-vessel in pericementum.

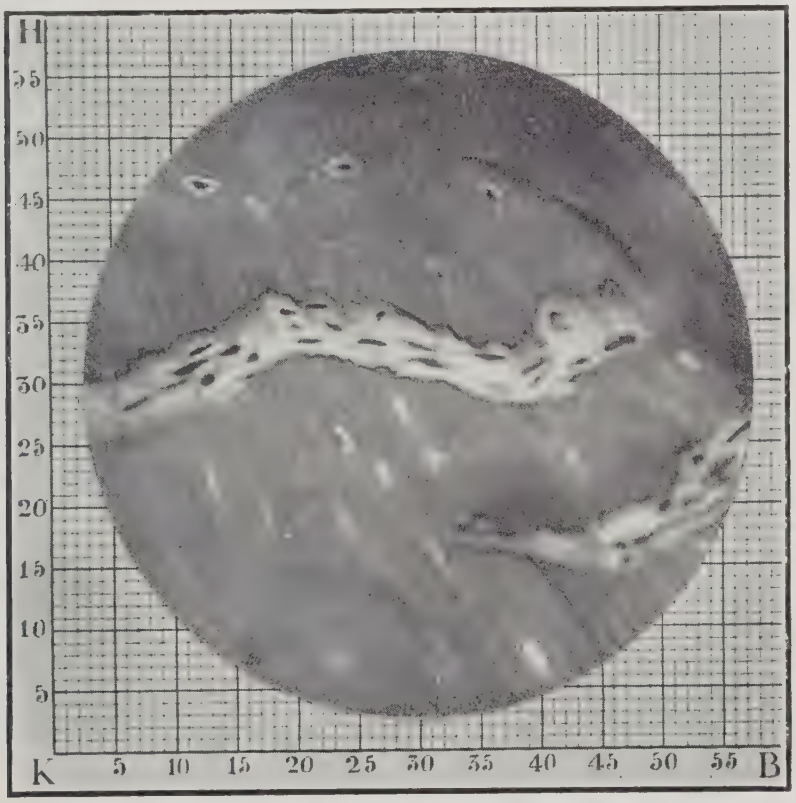


Fig. 68— x 500. Rarefying Pericementitis Fibrosa. Alveolar rarefaction. Perforating canals.

H K 35 x K B 18 } Perforating canals penetrating alveolar bone transversely to the lamellae.
H K 18 x K B 48 }
H K 33 x K B 31 New tissue.
H K 37 x K B 47, Bone-cell and lacuna undergoing absorption.

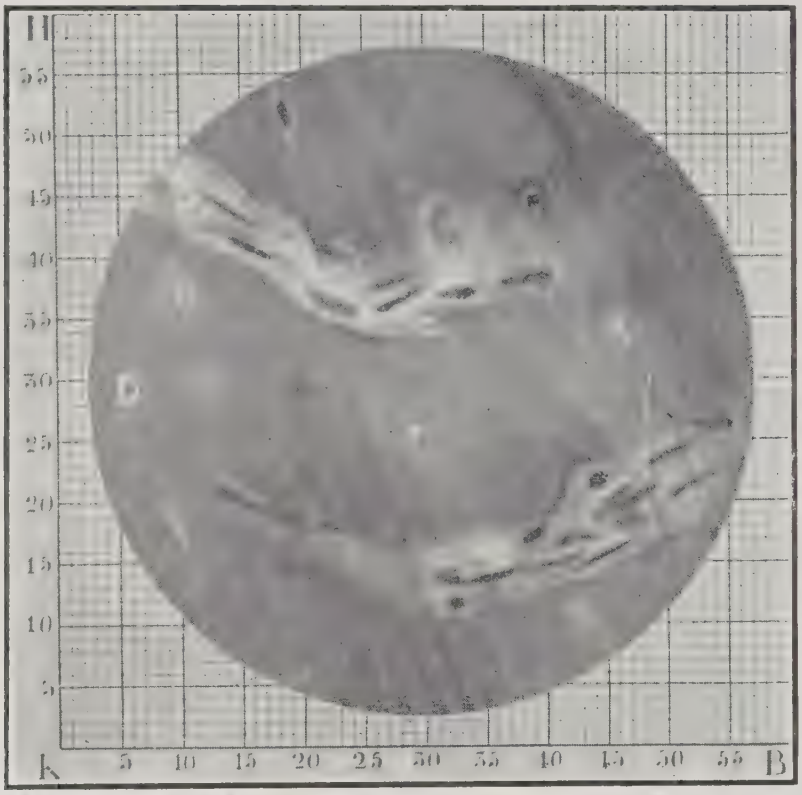


Fig. 69— x 750. Rarefying Pericementitis Fibrosa. Alveolar rarefaction. Perforating canals.

H K 43 x K B 36 } Terminations of perforating canals.
H K 20 x K B 16 }
H K 38 x K B 28, New tissue.
H K 45 x K B 39, Bone-cell and lacuna undergoing absorption.

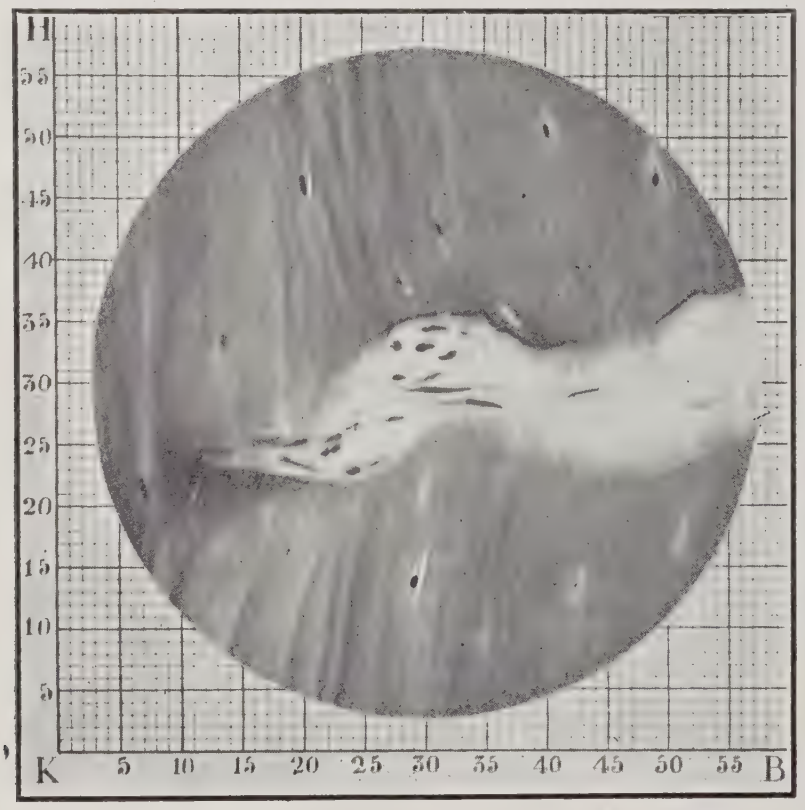


Fig. 70— x 500. Rarefying Pericementitis Fibrosa. Alveolar rarefaction. Perforating canal.

H K 30 x K B 40, Perforating canal.
H K 29 x K B 32, New tissue.
H K 35 x K B 31, Osteoblast.
H K 14 x K B 29, Bone-cell in lacuna.

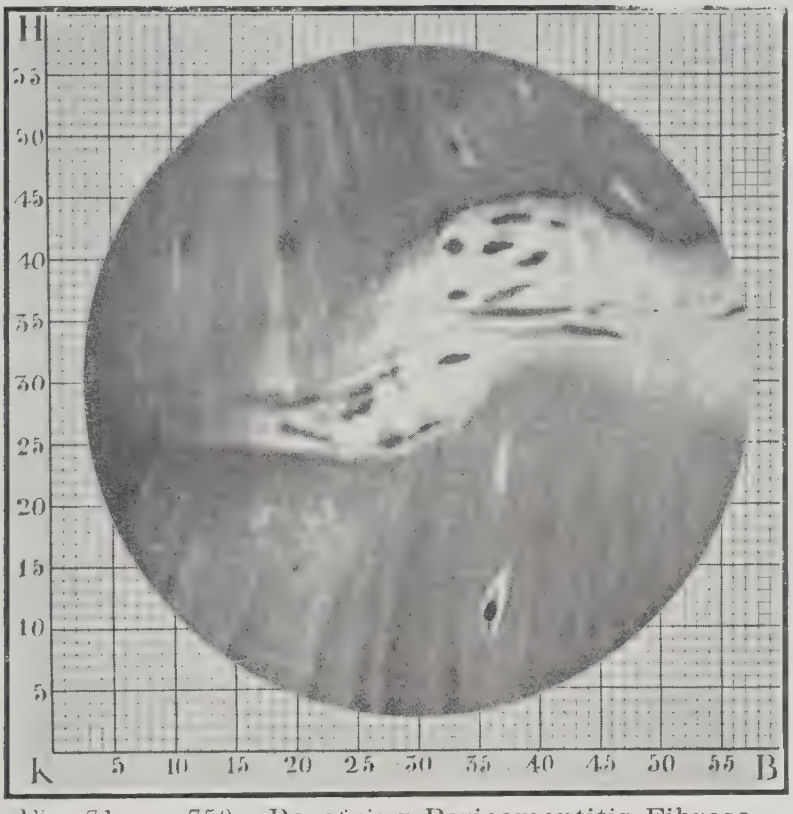


Fig. 71— x 750. Rarefying Pericementitis Fibrosa. Alveolar rarefaction. Perforating canal. High magnification of the specimen shown in the previous illustration.

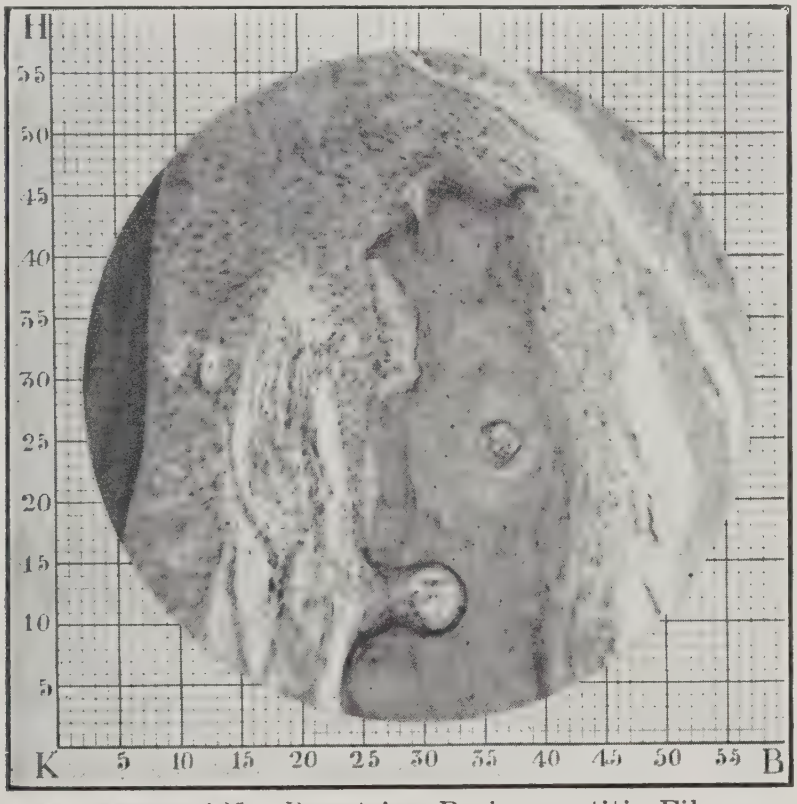


Fig. 72— x 125. Rarefying Pericementitis Fibrosa. Alveolar rarefaction. Nutritional atrophy.

H K 47 x K B 30, Zone of osteoid tissue at the crest of the alveolar.
H K 35 x K B 28 } Osteoid zones along the inner wall of the alveolar bone.
H K 22 x K B 25 }
H K 25 x K B 37, Haversian canal.
H K 30 x K B 7, Cementum.
H K 45 x K B 20, Pericementum.

In addition to these typical phases, and in frequent and varied association with them, there are certain zones in which the bone changes its character to an osteoid tissue. These zones are found on the surface of the bone and in the Haversian canals. The bony structure stains unequally, dependent upon the amount of constituent elements in the matrix. Pale pink-staining matrix takes the place of the calcified tissue, which is blue in its reaction with hematoxylin, and there is a loss of the bone-cells. These atrophic changes appear to be associated with nutritional disturbances caused by the new tissue (Figs. 72, 73, 74, 75, 76).

Other osteoid zones are to be noted, and they are regarded as regions showing bone-formation, in which the ground substance laid down by the osteoblasts is, as yet, calcium-free.

A minor phase of the rarefying process is the thinning of the bony lamellae by the coalescence of pit-like depressions, Howship's lacunae, which have been formed by the action of the multinuclear giant-cells, the so-called osteoclasts (Figs. 77, 78, 79, 80, 81).

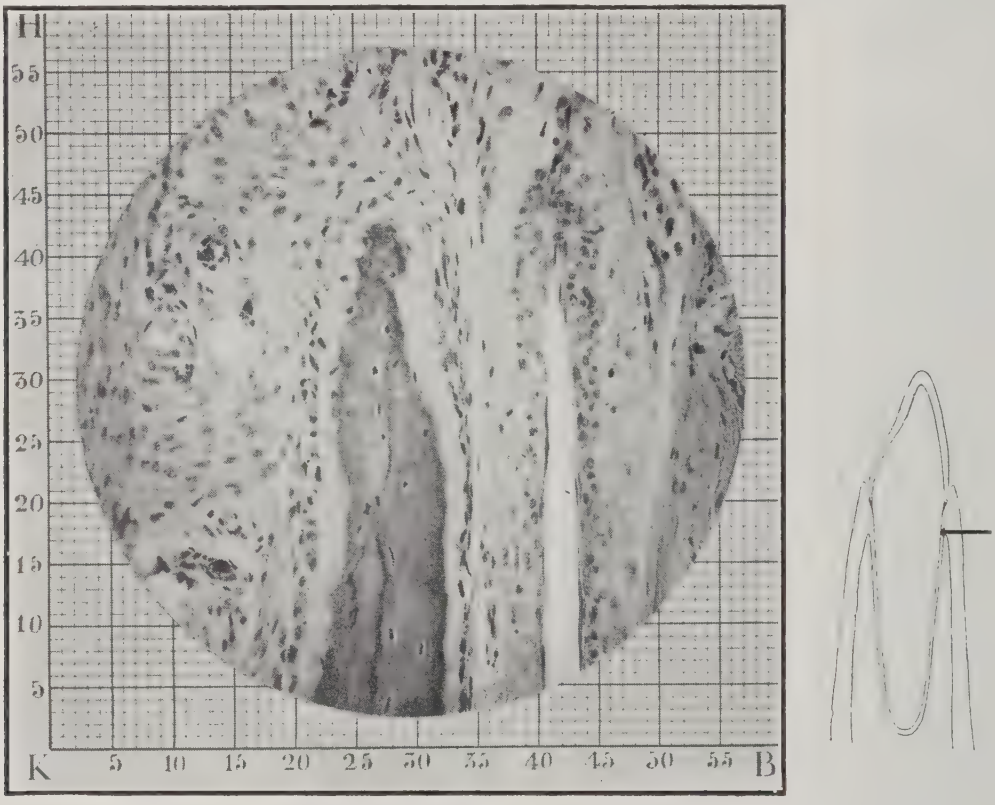


Fig. 73— x 375. Rarefying Pericementitis Fibrosa. Alveolar Rarefaction. Atrophy due to nutritional disturbance caused by the new tissue.

H K 45 x K B 28, Crest of alveolar process.
H K 5 x K B 20 } Borders of the alveolar process.
H K 3 x K B 34 }
H K 35 x K B 30 }
H K 44 x K B 28 } Osteoid tissue bordering the alveolar crest.
H K 19 x K B 23 }
H K 32 x K B 22, Border of osteoid tissue.
H K 32 x K B 24, Border of calcified tissue.
H K 32 x K B 29, Border of calcified tissue.
H K 32 x K B 32, Border of osteoid tissue.
H K 17 x K B 15, Mass of new tissue in pericementum.

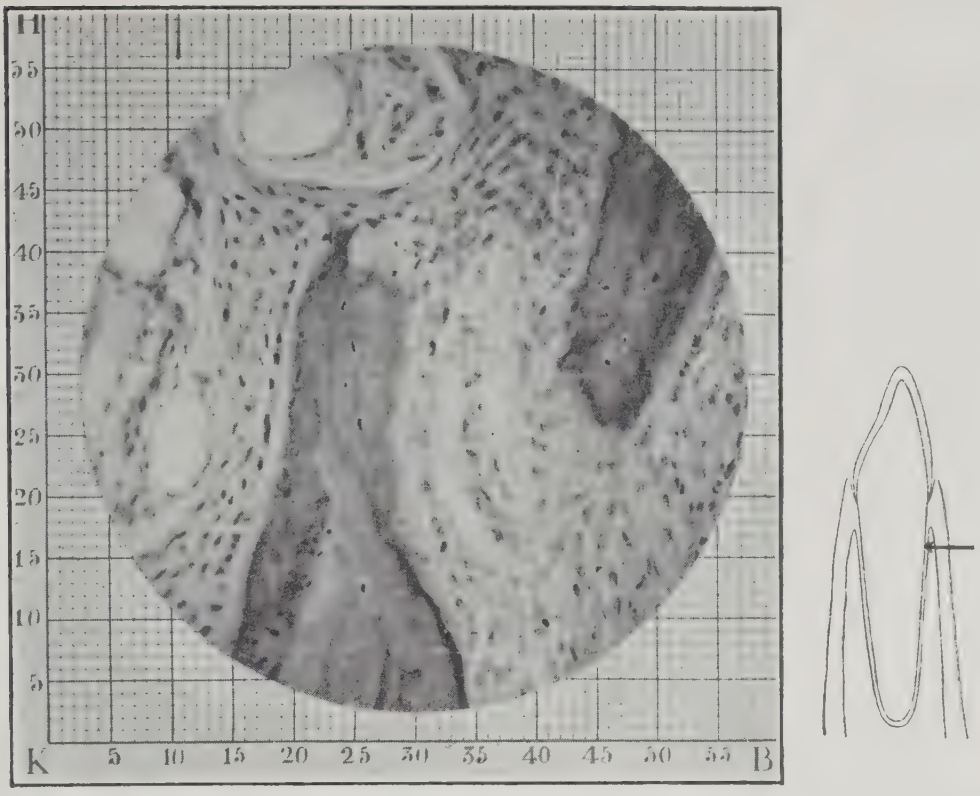


Fig. 74— x 375. Rarefying Pericementitis Fibrosa. Alveolar Rarefaction. Nutritional atrophy.

H K 43 x K B 25, Tip of segment of alveolar bone.
H K 40 x K B 27 } Osteoid tissue.
H K 18 x K B 30 }
H K 50 x K B 30 } Masses of new tissue in the pericementum.
H K 20 x K B 10 }
H K 25 x K B 45, Base of a segment of alveolar bone.

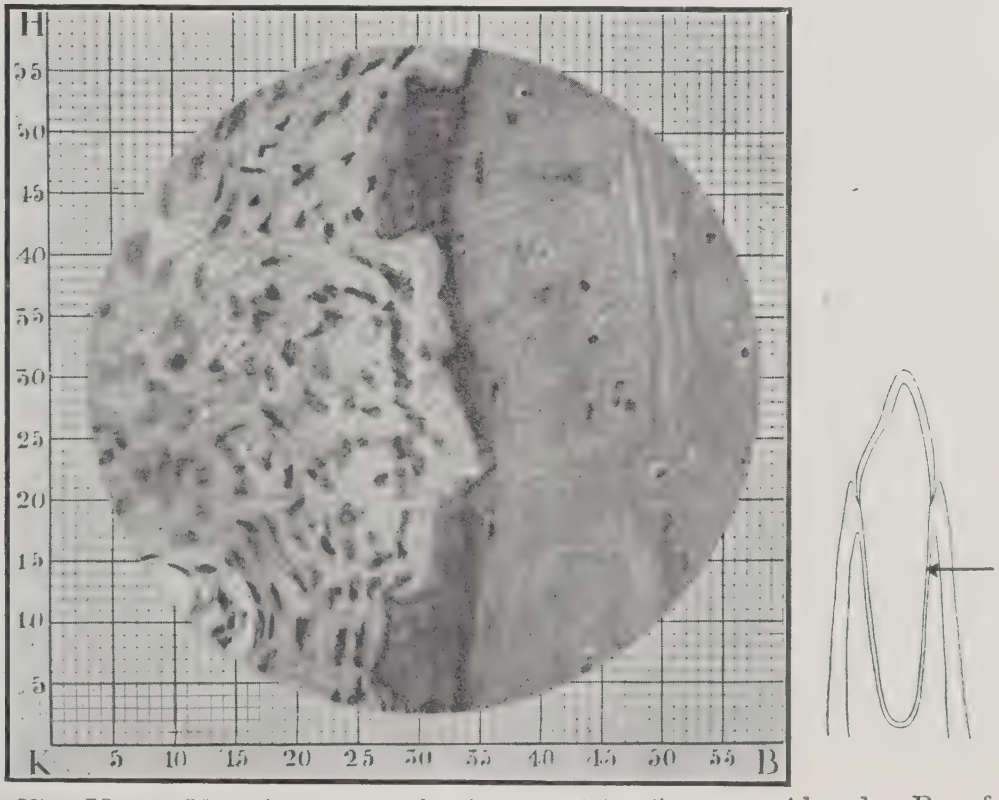


Fig. 75— x 500. Rarefying Pericementitis Fibrosa. Alveolar Rarefaction. Nutritional atrophy.

H K 28 x K B 32, Osteoid zone along the inner wall of the alveolar process.

H K 35 x K B 29, Row of osteoblasts along the border of the calcium-free zone.

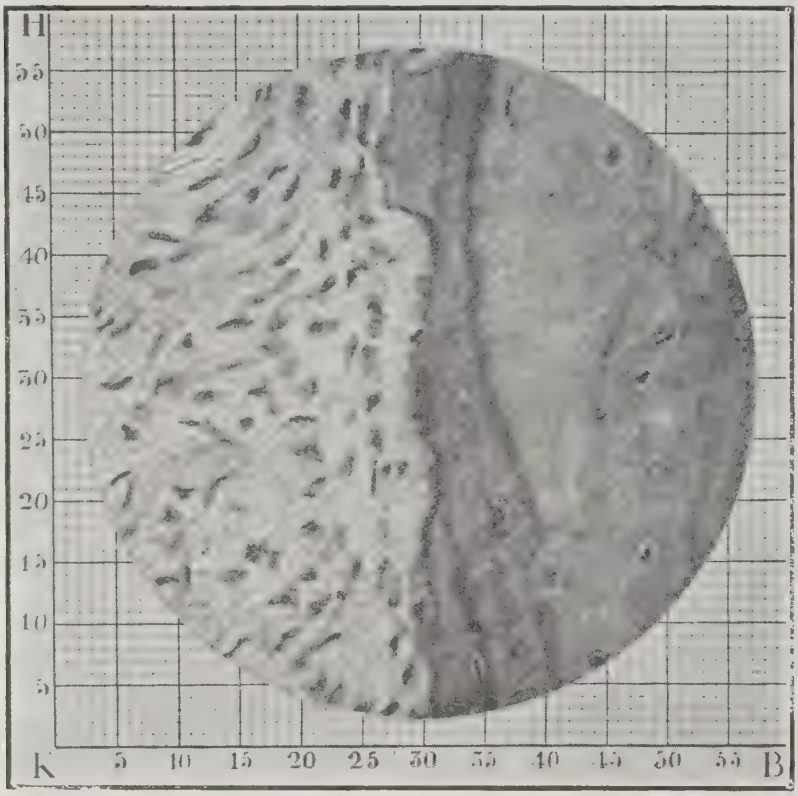


Fig. 76— x 750. Rarefying Pericementitis Fibrosa. Alveolar Rarefaction. Osteoid zone along the inner surface of the alveolar process.

H K 38 x K B 29, Osteoid zone. H K 34 x K B 25, Row of osteoblasts on the border of the calcium-free bone.

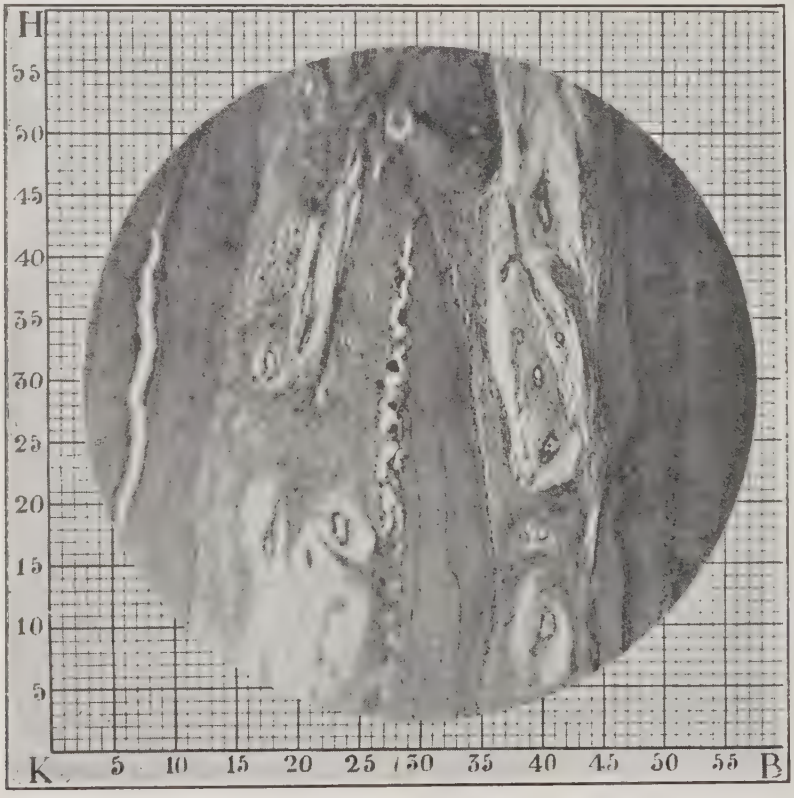


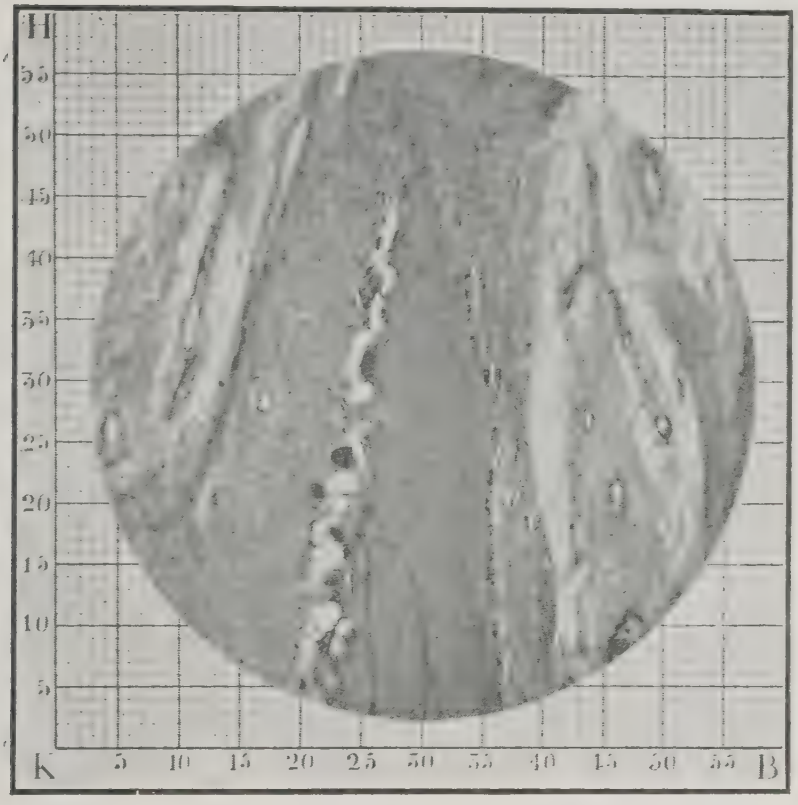
Fig. 77— x 94. Rarefying Pericementitis Fibrosa. Alveolar Rarefaction. Osteoclastic absorption. This section shows the bone in the bifurcation of a lower molar.

H K 45 x K B 30, Crest of alveolar bone.

H K 31 x K B 28, Osteoclast in Howship's lacuna.

H K 30 x K B 23 } Pericementum.
H K 30 x K B 40 }

H K 30 x K B 12 } Cementum.
H K 30 x K B 45 }



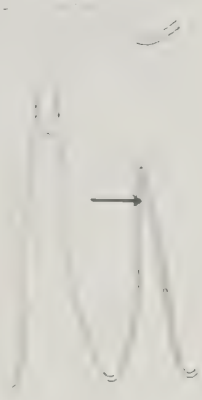
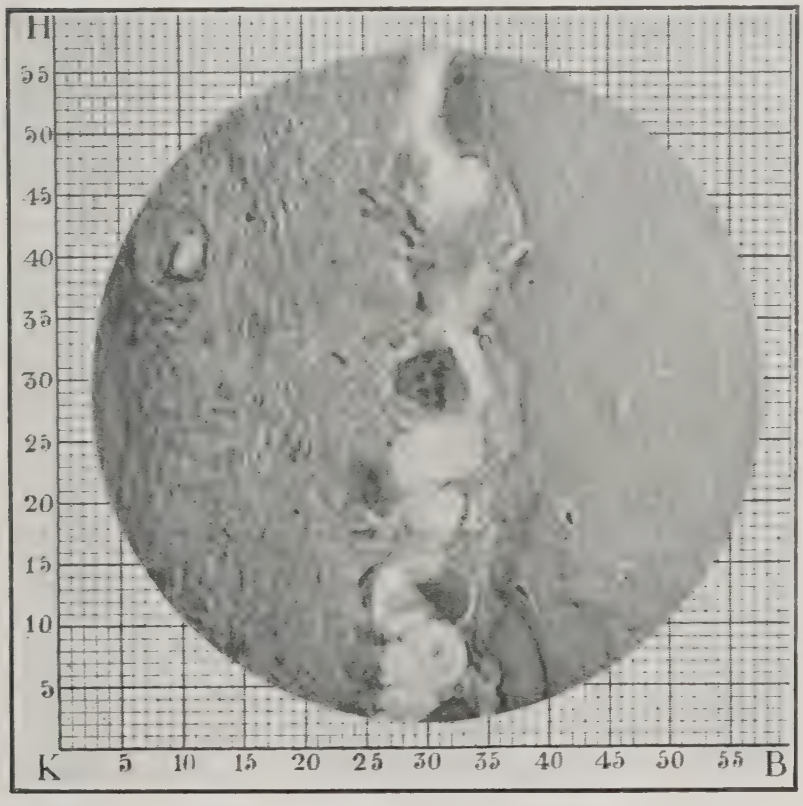


Fig. 78— x 188. Rarefying Pericementitis Fibrosa. Alveolar Rarefaction. Osteoclastic absorption. Higher magnification of the specimen shown in the preceding illustration.

H K 24 x K B 23, Osteoclast.
H K 47 x K B 30, Crest of alveolar process.
H K 23 x K B 35, Row of osteoblasts.



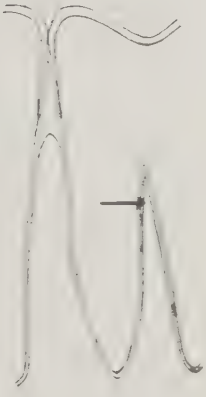


Fig. 79— x 500. Rarefying Pericementitis Fibrosa. Alveolar Rarefaction. Osteoclastic absorption.

H K 30 x K B 30, Osteoclast.
H K 28 x K B 37, Howship's lacuna.

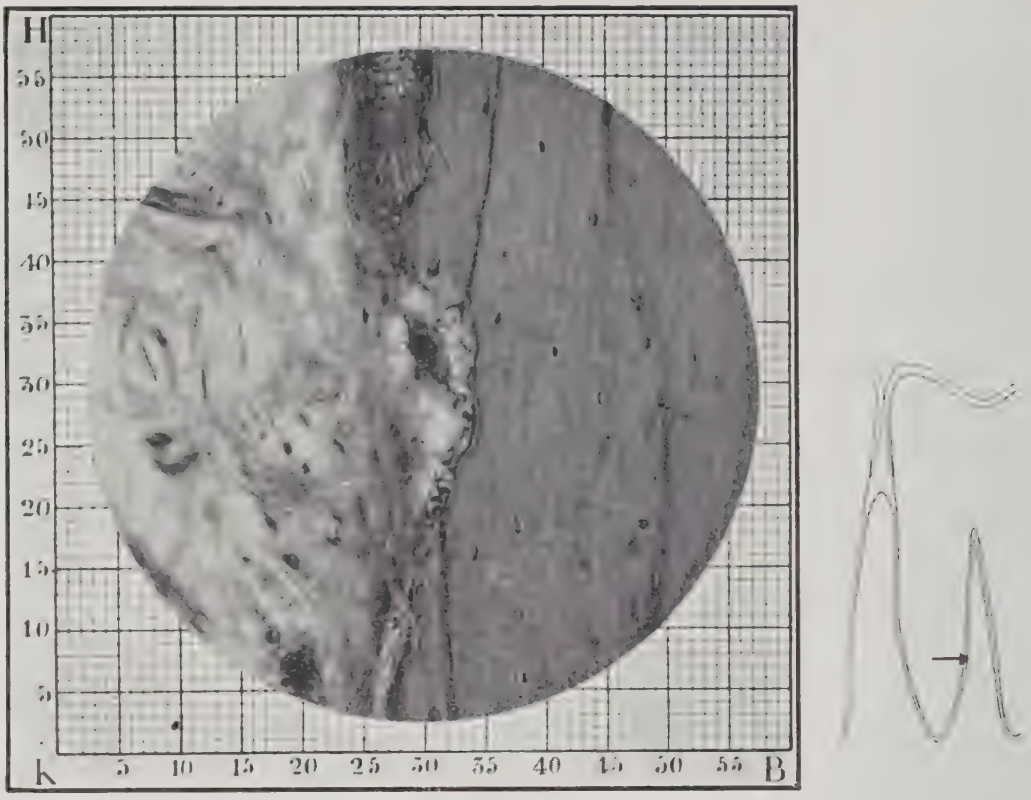


Fig. 80— x 500. Rarefying Pericementitis Fibrosa. Alveolar Rarefaction. Osteoclastic absorption.

H K 34 x K B 30, Osteoclast. H K 32 x K B 34, Howship's lacuna.
H K 30 x K B 15, Pericementum.

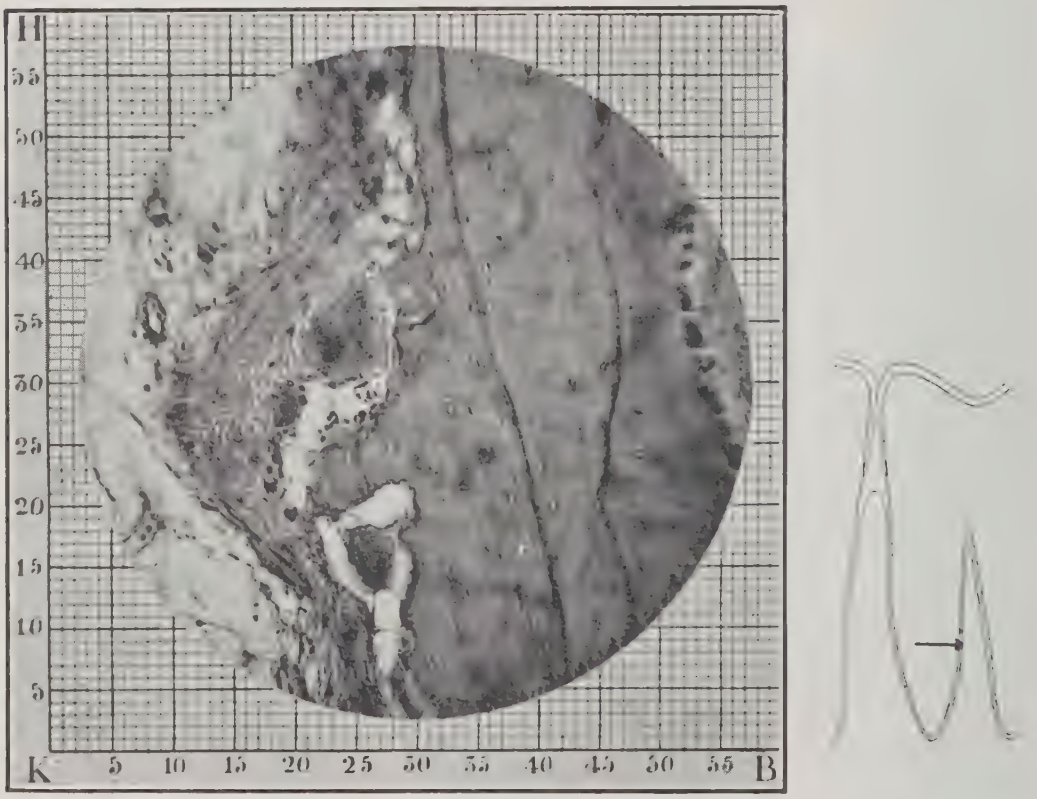


Fig. 81— x 375. Rarefying Pericementitis Fibrosa. Alveolar Rarefaction. Osteoclastic absorption.

H K 33 x K B 24 } Osteoclasts.
H K 16 x K B 26 }
H K 30 x K B 15, Pericementum.

Following upon these peculiar and characteristic changes taking place upon the surface of the bony trabeculae, wherein a specific type of reaction leads to the development of perivascular nodules, as well as of excavations of the bone, there develops a new tissue essentially composed of fibrous connective tissue, and which tends to replace the normal structure of the bone marrow. This fibrosis is laid down in an orderly fashion, and, during its growth, is not accompanied by the cells of the inflammatory exudate, nor by new capillaries such as are found in the development of true granulation tissue. The fibrous tissue, which appears in the marrow-spaces, develops as a hyperplasia, or it may be in response to a very mild and unknown stimulus, providing a proliferation of this tissue without inducing characteristic changes in the other structures. This fibrous tissue growth is so gradual and uniform that some have been inclined to regard it, in relation to disease of bone in other parts, as a type of reaction lying between inflammation, on the one hand, and new-growth, on the other. Much comment has been made in the literature concerning Osteitis Fibrosa of the long bones. In our studies, a similar reaction was found in the alveolar process, as well as in the pericementum, which we believe must be studied in parallel relation to that of other bones. Figs. 82, 83, 84.

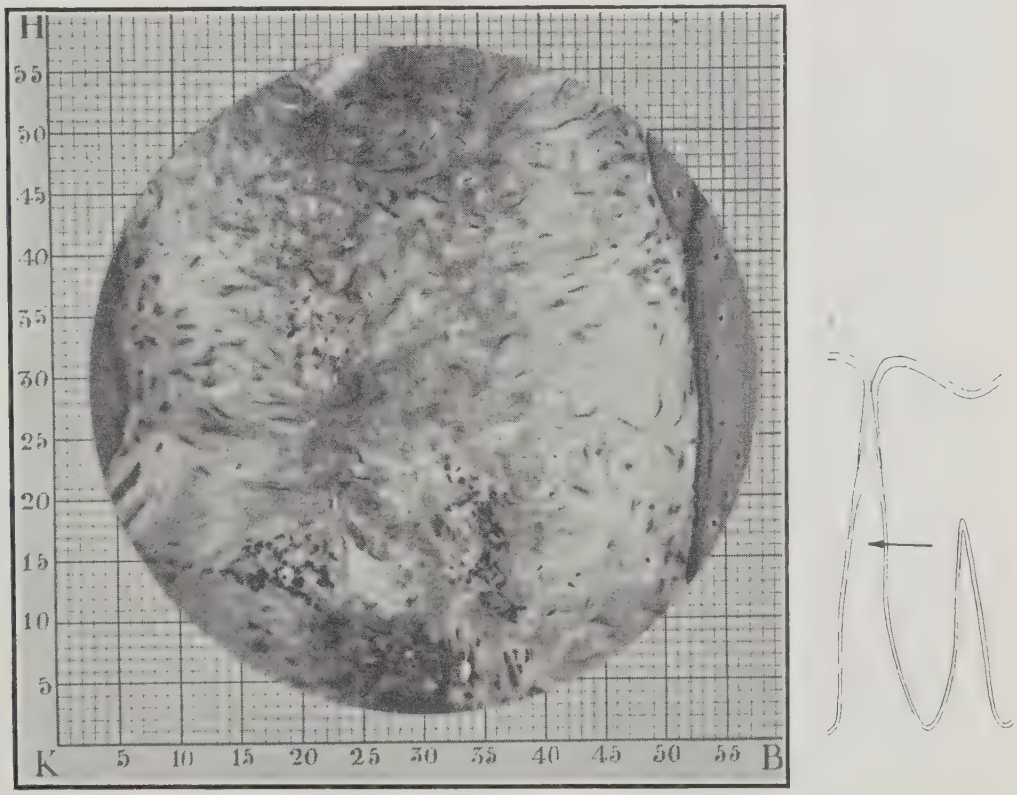
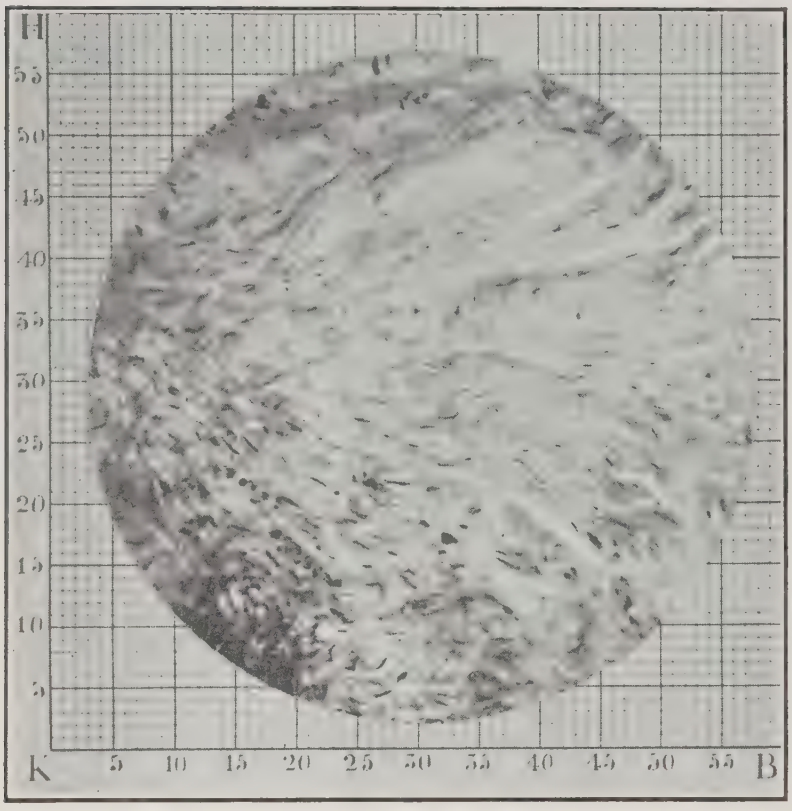


Fig. 82—x 375. Rarefying Pericementitis Fibrosa. Fibrosis of marrow-space.

H K 30 x K B 5 } Alveolar bone.
H K 30 x K B 55 }
Note numerous fibroblasts in intervening soft tissue.



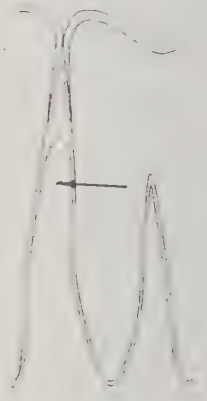
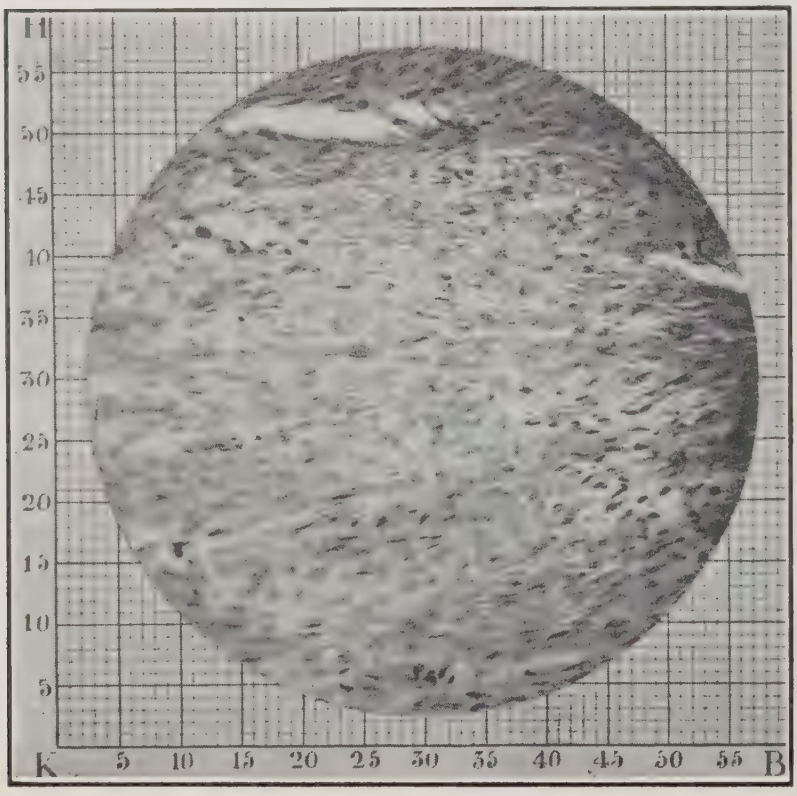


Fig. 83— x 375. Rarefying Pericementitis Fibrosa. Fibrosis at alveolar-margin.

H K 25 x K B 40 } Bundles of connective-tissue fibres.
H K 42 x K B 45 }



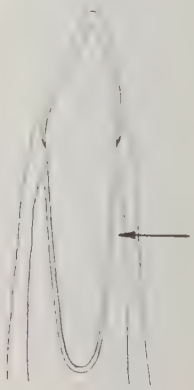


Fig. 84— x 375. Rarefying Pericementitis Fibrosa. Fibrosis subsequent to absorption of alveolar bone.

H K 32 x K B 35 Connective-tissue fibres.
H K 10 x K B 40

In the disease, Rarefying Pericementitis Fibrosa, the inflammatory changes noted in the gingival tissues are found to be extremely superficial, and they bear no relation to the changes that are observed in the pericementum. Furthermore, it is to be recognized that a varying infiltration by lymphocytes and plasma cells is constantly encountered in *normal* conditions in the mouth and surrounding parts. These inflammatory reactions vary from time to time, and seem to be found frequently in response to mild superficial irritations from foodstuffs and temperature changes.

The lesions of Rarefying Pericementitis Fibrosa localize themselves most often in the periodontal tissues of teeth which are in traumatic occlusion, or over-function, and appear to originate in pericemental tissues which are frequently subjected to abnormal occlusal stress.

Any tooth under occlusal stress, and acted upon by forces in equilibrium, presents to us the ideal picture of physiological occlusion. While physiological occlusion is based upon a mechanical equilibrium, that is, a condition in which the force of resistance offered by the alveolar process is equal to the occlusal stress directed through the tooth, the force of resistance maintained by the alveolar bone is probably dependent upon a state of equilibrium existing between the calcium content of the bone and the calcium content of the blood. It has been demonstrated by Wells that the calcium salts in bone are in a condition of flux, and that, if the body requires increased calcium, as during pregnancy, these calcium salts can be drawn upon. While occlusal traumatism seems to serve as the main factor in the localization of the lesions of Rarefying Pericementitis Fibrosa, certain conditions, in which the body is being robbed of its normal supply of calcium, seem, from a clinical standpoint, to accelerate the process of rarefaction.

When Rarefying Pericementitis Fibrosa is manifested as a pure type of periodontal disease, the phenomenon is usually observed in which there is a parallelism between alveolar resorption and gingival recession; in other words, recession of the gingival tissue making parallel progress with the bone resorption. The parallelism may be disturbed, however, by the establishment of infection in the gingival tissues. With the evolution of a superimposed, infective periodontitis, through consequent pocket formation, there is brought about the pathological composite, Chronic Periodontitis, Complex, to be described later. And there is much evidence to support the viewpoint that in the production of this infective periodontitis, of paramount importance as a causative factor, is the primary establishment of the disease, Rarefying Pericementitis Fibrosa.

The typical sites in the alveolar process of the rarefying phase of this disease are as follows: First, the parietal wall of the alveolus;

second, the crests of the alveolar process, including the process between the roots of bicuspids and molars; third, the crests, together with the periapical bone (the tooth situated in its alveolus furnishes a good example, in many instances, of a lever of the second class); and, fourth, the periapical bone.

The importance of early diagnosis of Rarefying Pericementitis Fibrosa cannot be over-emphasized. The primary lesion consists in the formation in the pericementum, of a new and pulp-like tissue, which replaces the normal pericemental structures, with interference of their normal functions. This is often manifested clinically by a slightly increased mobility of the tooth in its alveolus, due probably to the peculiar yielding character of the new tissue. The process of rarefaction, initiated by the new tissue, is frequently first manifested clinically by an increased radiolucency of the alveolar crest and associated osseous tissue. In other words, the crests of the alveolar bone become less impervious to the X-rays. This is followed by a change in the contour of these crests and a disturbance of their normal position, as related to the gingival line—the amelo-cemental junction. It is unsafe, however, to place entire dependence on the radiograph, since labial crest absorption may take place without registering on the X-ray negative. In such a case, careful examination of the relationship existing between the margin of the gingival tissue and the amelo-cemental line should be made. In the average normal case, the margin of the gingiva is approximately two millimeters above the amelo-cemental line. Also, the crest of the alveolar process in the average normal case is approximately two millimeters below this fixed landmark. Recession of the marginal gingiva is usually indicative of alveolar crest resorption. For example, a recession of the marginal gingiva which just exposes the amelo-cemental line will probably indicate a crest resorption of two millimeters. Exposure of the cementum will indicate an increased alveolar resorption of a corresponding degree.

The rarefying process frequently becomes initiated in the inner wall of the alveolar bone. In these instances, it is first manifested, radiographically, in certain zones, by an increased radiolucency of the lamina dura. Through progression of the process of rarefaction, by extension of the new tissue, the lamina dura is destroyed This alveolar loss is usually apparent in the radiograph, and there is thus exhibited a definite widening of the normal pericemental space. Through consequent thickening of the soft tissue content between the tooth and the bone, there is a further increase in the mobility of the tooth in its alveolus. Divergence from normal mobility, being dependent, as a rule, upon profound structural changes in the pericementum and alveolar bone, must be regarded as a diagnostic sign of the first importance.

PART IV: PERIODONTITIS.

The term periodontium is used to designate the supporting tissues of the teeth. The three essential tissue-components of the periodontium are, the gingivae, the pericementum, and the alveolar process. By the term periodontitis, is denoted an inflammation of the periodontium and a disease so designated involves all three components. On this distinct basis, the term makes for simplification and accuracy in terminology. It excludes certain lesions of the periapical tissues, such as abscesses and granulomata, because, while the pericementum and alveolar process are involved in these lesions, the gingivae are not.

Based on clinical and histo-pathological findings, inflammation of the periodontium may be classified as follows:

1. Acute Periodontitis (fuso-spirillary)
2. Chronic Periodontitis, Simplex { with pocket / without pocket
   Complex { with pocket / without pocket

From pathological and clinical viewpoints, Chronic Periodontitis, of the pocket-producing types, may be divided, as seen in the foregoing classification, into two main divisions, first, the Simplex, and second, the Complex. Either type of Chronic Periodontitis, however, is the resultant of certain preceding lesions which, by their coalesence, constitute a distinct pathologic composite.

The order in which these lesions occur in the mature form of the Simplex type is as follows:

1. Chronic Gingivitis, Cemental (pocket in cemental gingiva).
2. Infective Osteitis (Rarefying and Condensing).
3. Chronic Pericementitis (pocket extension into the pericementum).

In the mature form of the Complex Periodontitis there are to be found two principal lesions:

1. Rarefying Pericementitis Fibrosa.
2. Infective Periodontitis.

Infective Periodontitis is in its turn made up of three constituent lesions which have appeared as phases in the following order:

A. Chronic Gingivitis Cemental (pocket in cemental gingiva).
B. Chronic Pericementitis (pocket extension into the pericementum).
C. Infective Osteitis (Rarefying and Condensing).

The pocket which is exhibited in Chronic Gingivitis, Cemental, and Chronic Pericementitis, may be produced in two entirely different ways, although its histo-pathology is essentially the same in both types. Chronic Pericementitis is a common pathological factor in both the Simplex and Complex types of Periodontitis and manifests the same sequence of pathological changes.

In the Simplex type, the gingival lesion is the dominating one, and is not so extensive or rapid in pocket development as in the Complex type. It represents a simple reaction to bacterial invasion, whereby, through local lowered resistance in the gingival tissues, infection has been established. Consequent upon the loss of gingival attachment, absorption of the alveolar crest takes place, again an inflammatory reaction to toxic agents. In the majority of cases in which superficial causes are the dominating ones, progress is fairly slow, because of the fact that the pericementum is high in resistance.

In the Complex type of Chronic Periodontitis the formation of the pocket is a phenomenon preceded by much more complicated and profound changes than occur in a simple gingivitis. The ideal requirements for rapid and deep pocket extension are alveolar resorption, lowered resistance in part or all of the pericementum, and the establishment of gingivitis. Disturbance of equilibrium, as manifested in excessive function or traumatic occlusion, while but one etiological factor in the production of the required changes in the pericementum and alveolar process, is the most important one.

In the Periodontitis, Simplex, which, in its mature state, simulates the Complex, the progression is centripetal, while in the Complex type, the course is in the main, centrifugal. In other words, in both the Simplex and Complex types, the inflammation, subsequent to the establishment of infection, progresses *from without inwards.* But in the Complex type, the establishment of infection is subordinate to the lesion Rarefying Pericementitis Fibrosa, which has, as a rule, preceded it. By the fact of this sequence the indication is that the pathologic progression in the Complex Periodontitis is, in the early stages, *from within outward.*

HISTO-PATHOLOGY OF THE BASIC LESIONS OF CHRONIC PERIODONTITIS, COMPLEX.

RAREFYING PERICEMENTITIS FIBROSA.

We have previously stated that Chronic Periodontits, Complex, is a disease produced through the coalescence of two distinct lesions, Rarefying Pericementitis Fibrosa and Infective Periodontitis. We have also stated that Infective Periodontitis is in its turn a composite of three lesions. We next proceed to a consideration of the nature of these lesions and their relationship to each other.

In stating that rarefaction of the alveolar process, as a rule, precedes gingivitis, in the type of periodontitis we choose to term Chronic Periodontitis, Complex, the researches of Hopewell-Smith, Roy, Gottlieb, Fleischmann and the writer have met on common ground.

These investigators state, however, that the primary lesion is a *marginal alveolar atrophy*, while our findings have led to the conclusion that the primary pathological change is *a chronic and progressive lesion of the pericementum*, which results in the rarefaction and destruction of the alveolar bone.

That true atrophic changes in the alveolar process can occur is not denied. Excellent examples can be seen about teeth that have become functionless during natural life. In other words, the enforced rest has brought about a true atrophy. In the mouth, a condition is frequently seen which is characterized by an entire absence of occlusal contact of certain teeth, from the time of their eruption, no matter what position the mandible assumes. This furnishes another example of lack of function, which brings about, sooner or later, atrophic changes in the periodontal tissues. These changes are dependent upon defective nutrition, as normal nutrition of the alveolar process and pericementum depends largely upon normal function of the teeth supported. Senile changes also depend, in part, upon defective nutrition of the tissues owing to changes in the blood-vessels which occur almost regularly with advanced age.

While insisting upon a most exact consideration of the histopathological findings the clinical observations of prominent periodontists cannot be overlooked. There is sufficient clinical evidence to prove that rarefaction of the alveolar process is almost constantly associated with over-functioning teeth. The splendid work of Stillman and McCall has brought to the attention of the profession, in a way not heretofore shown, the injurious effects of over-function upon the periodontal tissues, as manifested in traumatic occlusion. Traumatic occlusion seems to serve as the main factor in the localization of this rarefying process.

The type of rarefaction of the alveolar process, most commonly found, is a phase of a complex disease condition termed by the writer Rarefying Pericementitis Fibrosa. It is to be kept in mind that the important, though not the spectacular element in this disease, is the formation in the *pericementum* of a new tissue which has previously been described. The histo-pathology of this disease has been given in detail in an earlier part of this paper.

INFECTIVE PERIODONTITIS.

Infective Periodontitis, as previously stated, is a composite disease produced by the coalescence of Chronic Gingivitis (cemental), Chronic Pericementitis and Infective Osteitis. These three lesions

occur in a sequential relationship and may be regarded as phases in the development of Infective Periodontitis. The relationship between Rarefying Pericementitis Fibrosa and Infective Periodontitis is a very important one. Were it not for the primary establishment of Rarefying Pericementitis Fibrosa, the Infective Periodontitis would establish itself as a Chronic Periodontitis, Simplex, a disease characterized by slow progression, shallow pocket formation, and little divergence from normal mobility in the early stages.

The difference between Chronic Periodontitis, Simplex, and Chronic Periodontitis, Complex, as observed clinically is most spectacular. In the Complex type, the prominent characteristics are: rapid development of the periodontitis, the formation of a deep and narrow pocket, and marked divergence from normal mobility.

In this latter type, the Rarefying Pericementitis Fibrosa is not only the primary lesion, as regards pathological progression, but it is actually a causal lesion in the production of Infective Periodontitis, and having once brought about the establishment of Chronic Periodontitis, Complex, the Rarefying Pericementitis Fibrosa remains, in many cases, the dominating lesion of the composite. This lesion is causal in that, in part, it provides the requisites for the establishment of infection in the gingivae and its progression into the deeper structures.

PHASE 1. CHRONIC GINGIVITIS—*Cemental.*

Cemental-gingival pocket.

A cemental-gingival pocket is a lesion occurring at the base of the gingival crevice, whereby a certain amount of separation of the tissue of the cemental gingiva from the cementum has taken place.

The cemental gingiva is that division of the gingivae which rests upon and is supported by the crests of the alveolar process. It is attached to the cementum from the level of these crests to the amelocemental junction. This tissue in turn supports the marginal gingiva, which by its apposition with the enamel forms the gingival crevice. It will be recalled that its circulation is derived in part from the pericementum, in part from the alveolar mucosa. Its circulation is also continuous with that of the marginal gingiva. It is subject to circulatory disturbance arising in either the pericementum or marginal gingiva. This fact together with its proximity to the gingival crevice which so frequently contains pathogenic bacteria, renders this tissue peculiarly susceptible to infection.

When infection takes place in the cemental gingiva, it usually occurs at the base of the gingival crevice, where through imperfect junction of the epithelial cells with the cementum or because of the extreme thinness of this defensive covering, invasion of the underlying connective tissue by bacteria is readily accomplished. Under the

microscope, the tissue in this locality exhibits infiltration by plasma cells and lymphocytes, which seem to be the predominating leukocytes in this lesion. These infections are therefore, as a rule, of the sub-acute type.

Because of the fact that these lesions are usually initiated as subacute inflammations, the classic signs of acute inflammation are lacking. This most important pathological process is therefore extremely difficult to diagnose clinically, until sufficient separation from the cementum has occurred to permit of measurement by the probe.

It will be recalled that we are considering a process which is leading toward a separation of the tissue of the cemental gingiva from the cementum. This separation which creates the so-called pocket, results in a denudation of the cementum. A striking feature of this process is the role played by the epithelium at the base of the gingival crevice. This epithelium exhibits a very constant tendency to proliferation and extension apically. The pocket is usually formed in one of two ways. Either the epithelium extends for some distance along the cemental surface, and subsequently becomes separated from it (figs. 85, 86 and 87); or by a progression of the inflammatory process, ulceration is established, with a consequent denudation of the cementum. In the first instance, ulceration occurs, if at all, as a secondary phenomenon to

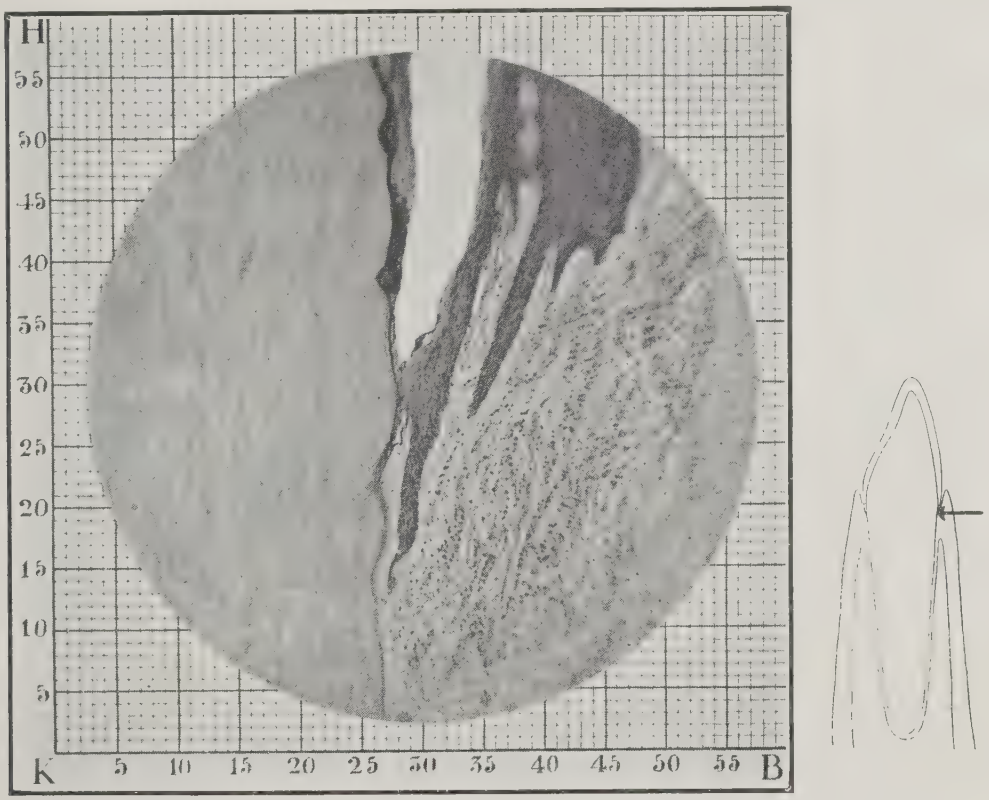
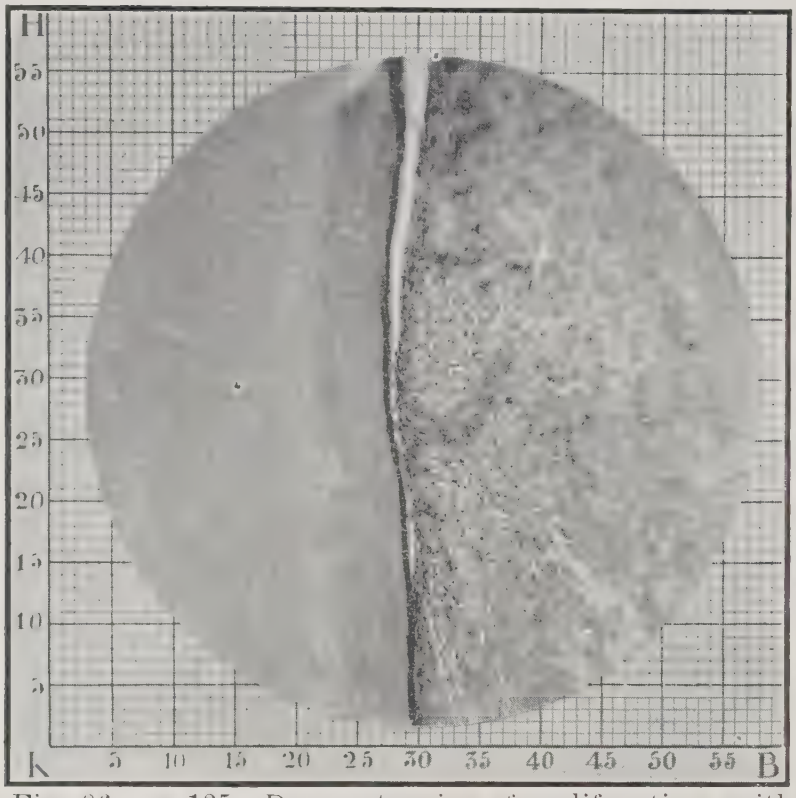


Fig. 85— x 125. Early proliferation and extension of crevicular epithelium at the base of the gingival crevice. Tissue change leading to the formation of a pocket in the cemental gingiva.

H K 32 x K B 28, Base of gingival crevice.
H K 19 x K B 29, Proliferating epithelium.
H K 19 x K B 35, Leukocytic infiltration.



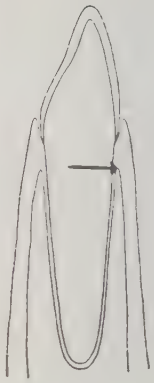


Fig. 86— x 125. Deep extension of proliferating epithelium along the cementum and beginning separation. Early pocket formation in the cemental gingiva.

H K 4 x K B 30, Extremity of proliferating epithelium.
H K 15 x K B 30, Base of newly-formed pocket.
H K 33 x K B 32, Leukocytic infiltration. Note that alveolar bone has been absorbed in advance of the extension of the pocket. The original position of the crest of the alveolar process was probably H K 14 x K B 36.

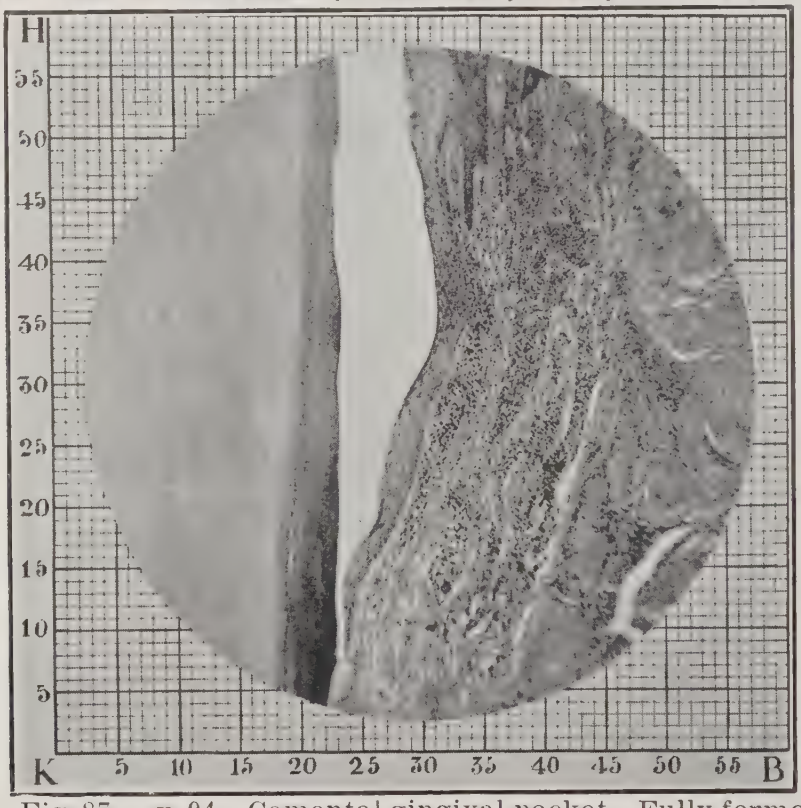


Fig 87— x 94. Cemental-gingival pocket. Fully-formed pocket, showing the epithelial covering of the detached soft tissues.

H K 9 x K B 23, Base of pocket.
H K 25 x K B 28, Epithelial covering of soft tissues.
H K 30 x K B 37, Leukocytic infiltration.
H K 20 x K B 23, Cemental surface.

the tissue separation. In the second instance, tissue separation takes place primarily through the ulcerative process (figs. 88 and 89). In this latter case, any proliferating epithelium at the base of the crevice will usually be included in the ulceration. Whether ulceration is primary or secondary in sequence, the tendency is for the process to become reduced to a chronic non-ulcerative inflammation, with the epithelium covering the separated soft tissue.

We have stated that the diagnosis of a cemental-gingival pocket is extremely difficult in its early stages. While this lesion may occasionally have an acute onset, evidenced by redness, swelling and tenderness to touch, this stage as a rule soon passes into the sub-acute stage. In the absence of the microscope, we have only the probe as a diagnostic aid. Regardless of whether there is a visible pus formation or not, a separation of soft tissue from the tooth which permits the insertion of a probe to a depth of more than two millimeters is, in the average case, indicative of pocket formation.

The distance from the amelo-cemental junction to the level of the alveolar crest, or in other words, the depth of the cemental gingiva, is on the average, two millimeters. The average depth of the gingival crevice on all surfaces is also two millimeters. It therefore follows that when the probe can be inserted under the gingival margin for a dis-

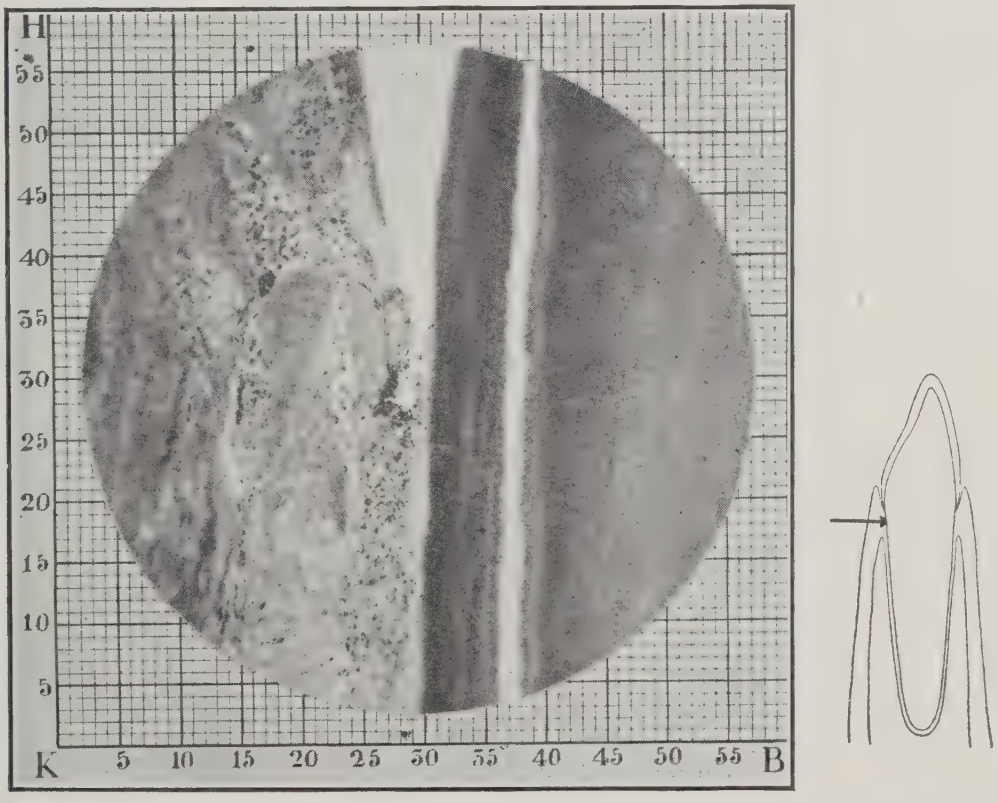


Fig. 88— x 187. Pocket formation through the extension of ulceration along the cemental surface. (This is a later stage of chronic ulcerative cemental gingivitis.)

H K 28 x K B 28, Base of ulcer.
H K 28 x K B 34. Cementum.

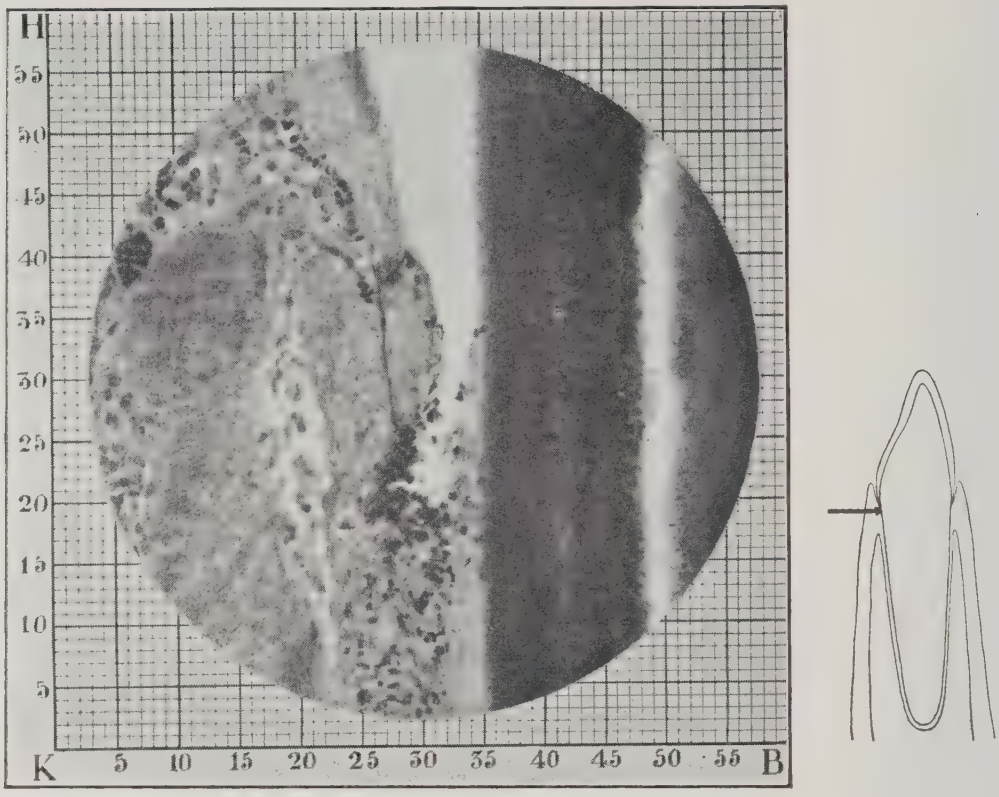


Fig. 89— x 375. Higher magnification of the specimen shown in the preceding illustration.

H K 21 x K B 30, Base of ulcer.
H K 51 x K B 27, Extremity of cervicular epithelium.
H K 7 x K B 29, Leukocytic infiltration. Note that pericemental tissue still persists above the deepest extension of the ulcerative process.

tance between two and four millimeters it indicates pocket formation of corresponding depth in the cemental gingiva. If it penetrates more than four millimeters, it may be assumed that the pocket involves the pericementum.

PHASE 2. CHRONIC PERICEMENTITIS.

Pericemental Pocket

A pericemental pocket is an extension of a cemental-gingival pocket into the pericementum. It will be recalled that the pericementum is that tissue which fills the space between the root of the tooth and the wall of the alveolus.

The pericementum is subject to circulatory disturbances due to interference with its normal function. It thus may become subject to infection which may extend into it from the cemental gingiva. Previous to infection of the pericementum by extension from the cemental gingiva either with or without pocket formation, the crest and associated alveolar process has become resorbed through the Rarefying Pericementitis Fibrosa, already described. The teaching of Black is that bone absorption occurs secondarily to the detachment of the fibres which had been originally inserted into it, and that

"detachment of the peridental membrane from the cementum is the principal factor in causing loss of the bone."[1] If bone absorption occurred in this sequence, it would necessitate the extension of the pocket between the tooth and the adjacent alveolar wall.

While bone absorption frequently takes place in such a way as to leave a considerable amount of bone above the base of the pocket, the significant fact is that the bone which constituted the original alveolar wall of the affected tooth is invariably resorbed in advance of the extending pocket. Furthermore, the bone which remains above the level of the base of the pocket, when viewed under the microscope, is seen to be so far removed from the affected tooth as to be well beyond the confines of the region in which the pocket was originally formed.

Examination of over six hundred specimens of pocket formation in its relationship to bone absorption has failed to reveal a single instance in which the pocket extends between the root of the tooth and the original alveolar wall.

The process by which a pocket is formed in the pericementum is identical with that which previously led to pocket formation in the cemental gingiva.

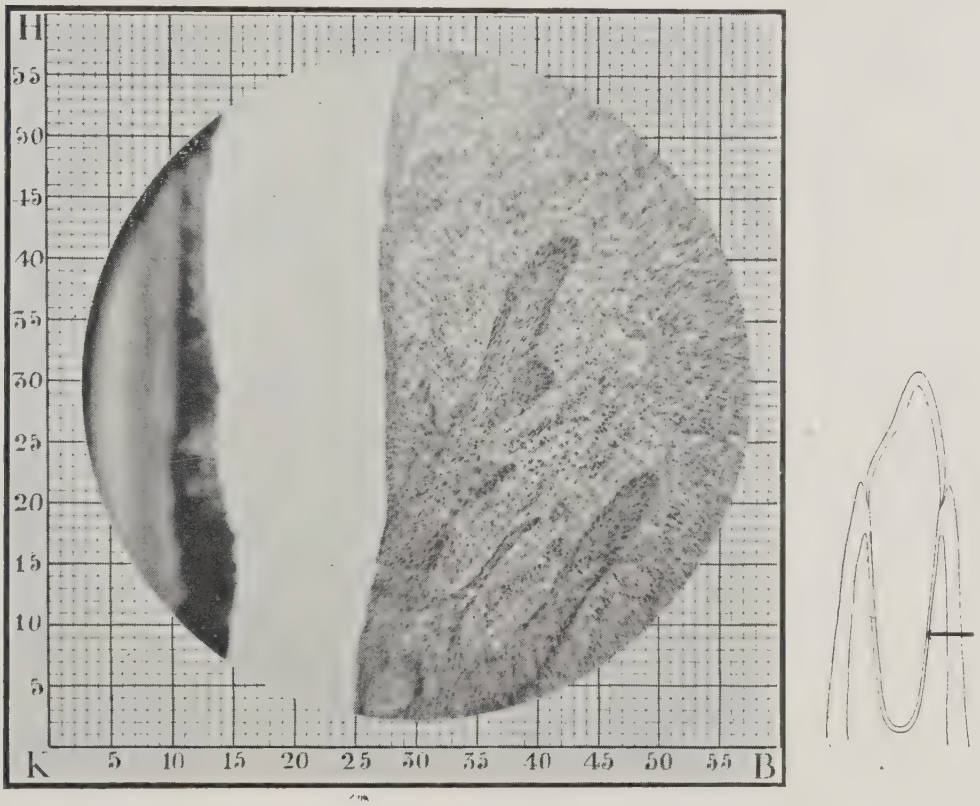


Fig. 90— x 125. Extension of the pocket into the pericementum.

H K 25 x K B 22, Pocket.
H K 20 x K B 30, Epithelial covering of detached soft tissues.
H K 15 x K B 13, Deposit of calculus in pocket.
H K 20 x K B 7, Cementum.
H K 25 x K B 44, Leukocytic infiltration. Note entire absence of alveolar bone in this section.

[1]Special Dental Pathology; G. V. Black, p. 170.

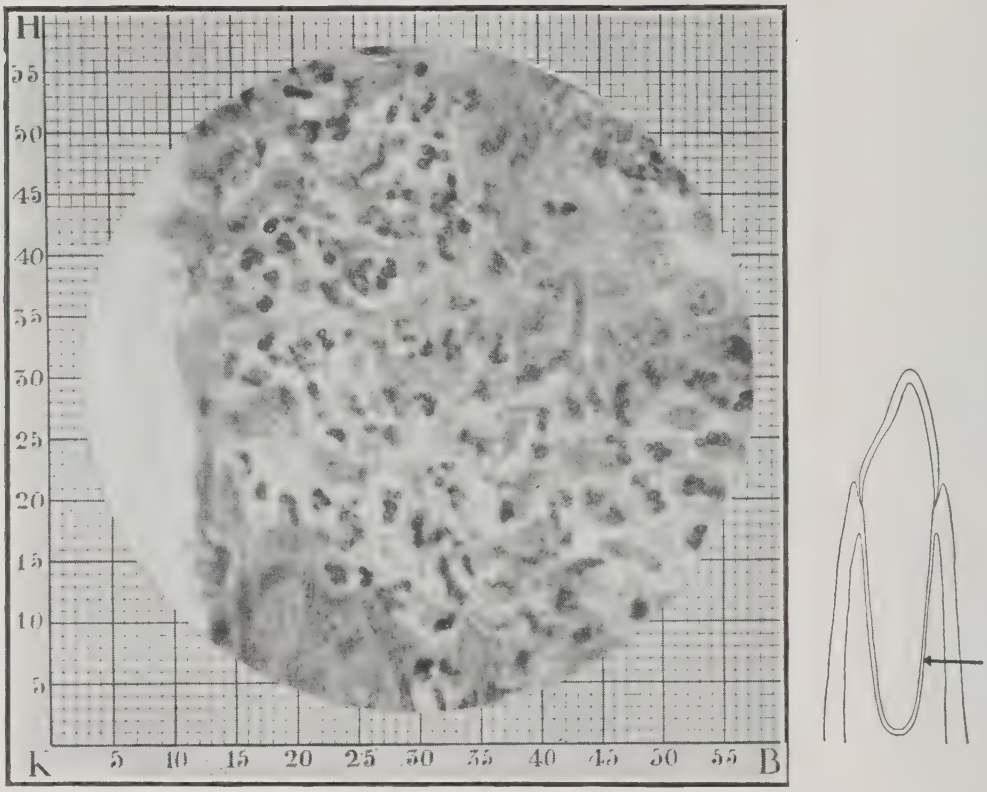


Fig. 91— x 1125. Very high magnification of the surface of the detached soft tissue shown in the preceding illustration. Note the intact epithelium on the surface, and the presence of polymorphonuclear leukocytes. The indications are that an inflammatory exudate of a purulent character is passing through the epithelial covering into the pocket. As yet there is no actual ulceration.

H K 30 x K B 6, Pocket.
H K 33 x K B 13, Epithelial covering of soft tissue.
H K 40 x K B 15
H K 33 x K B 30
H K 19 x K B 27
} Polymorphonuclear leukocytes.

Due to the progression of the inflammation, there is an active proliferation of the epithelium apically along the cementum. As in the case of cemental-gingival pocket formation, the pathologic process may exhibit either separation of the proliferating epithelium from the cementum, or an ulceration at the base of the advancing pocket. In either case, the significant fact is the constant tendency, and one which is successfully carried out in the majority of cases, for this epithelium to cover the separated connective tissue (figs. 90, 91, and 92). When a large number of specimens are examined it is surprising how little ulcerated tissue is to be found even in the deepest pockets, or in contact with large masses of calculus.

While pocket formation can and does take place through the stripping of the fibres of the pericementum by the extension of the process of ulceration, examination of hundreds of specimens showing pocket formation has forced the author to broaden his concept of the nature of this pathologic process. The cumulative evidence on this point emphasizes more and more the importance of the role played by

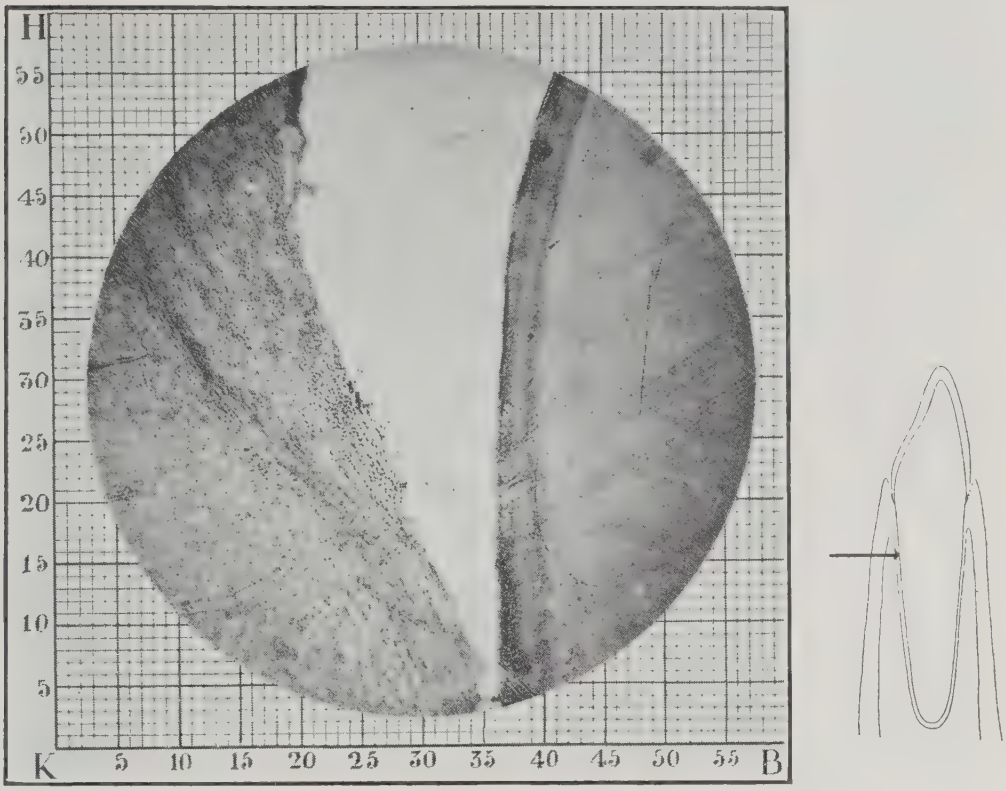
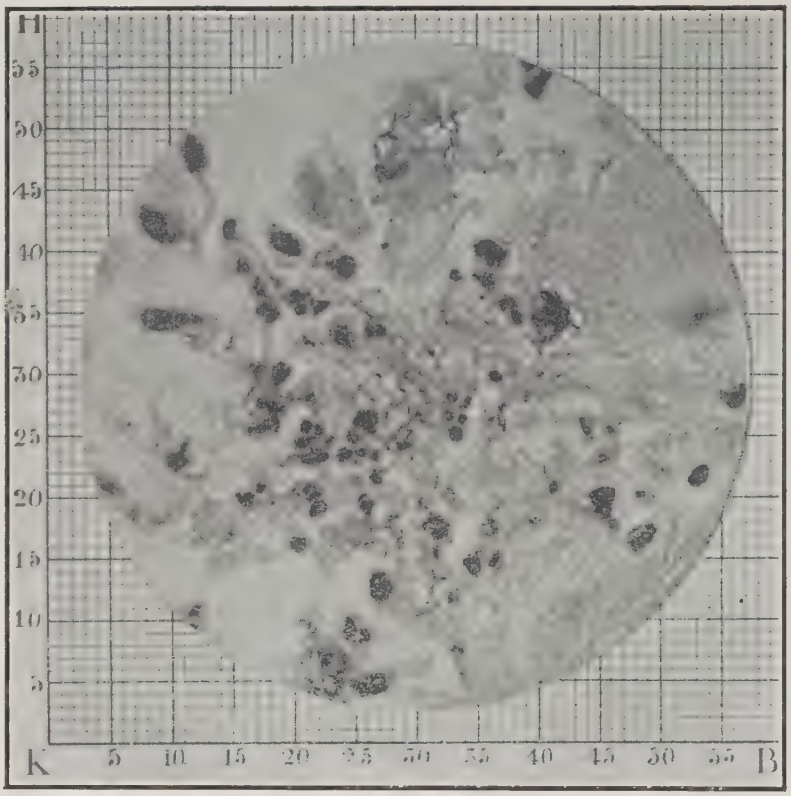


Fig. 92— x 94. Pericemental pocket. Extensive ulceration of the soft tissues of the cemental gingiva and pericementum.

The ulcerated surface extends from H K 51 x K B 21 (which was the point of original attachment of the crevicular epithelium) to the present base of the pocket.

epithelial proliferation in pocket formation. This evidence also tends to minimize the part played by ulceration as the important factor in the extension of the pocket. It thus becomes plain that our theories regarding treatment should be based on the assumption that in practically all pockets, the detached tissue sooner or later becomes covered with epithelium. In other words, there is a constant tendency, due to the defensive power of the organism, to reduce this disease process to a chronic non-ulcerative type of inflammation. It should also be noted that ulceration is not a prerequisite to a suppurative pericementitis, as the inflammatory exudate may pass from the tissues through the epithelium into the pocket. At times, due either to an increased virulence of the bacteria or a decreased local or general resistance, there may be exacerbations of acute inflammation, with increased purulent exudation, and other local manifestations (figs. 93, 94 and 95). At such times the inflammatory process may terminate in actual ulceration, which may persist for a considerable period of time. Due to inadequate drainage or other factors, acute abscesses may develop near the base of the pocket. These have been known clinically as pericemental abscesses.

The cementum underlying the pocket and from which the soft tissues have been detached always presents a septic surface. This is due to the frequent occurence of serumal deposits, to the retained



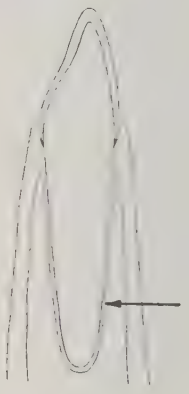


Fig. 93— x 1500. Chronic Pericementitis (Pocket in Pericementum). Bacteria in an ulcer at the border of a pericemental pocket.

H K 24 x K B 22 } Pus-cells (Dead polymorphonuclear leukocytes).
H K x 26 x K B 33 }
H K 21 x K B 53, Lymphocyte.
H K 27 x K B 28, In this region occur numerous bacilli.

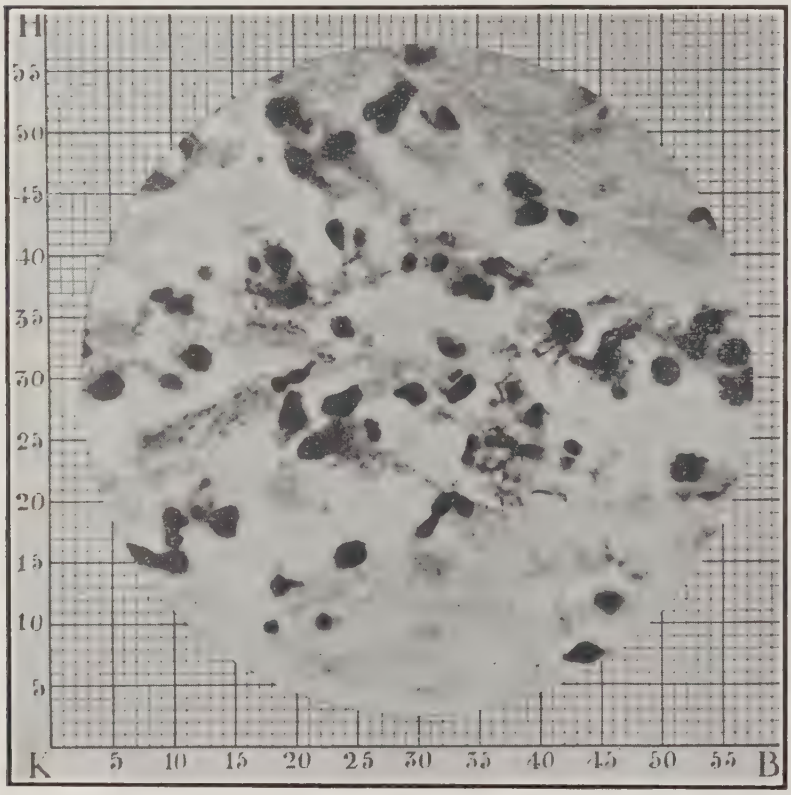
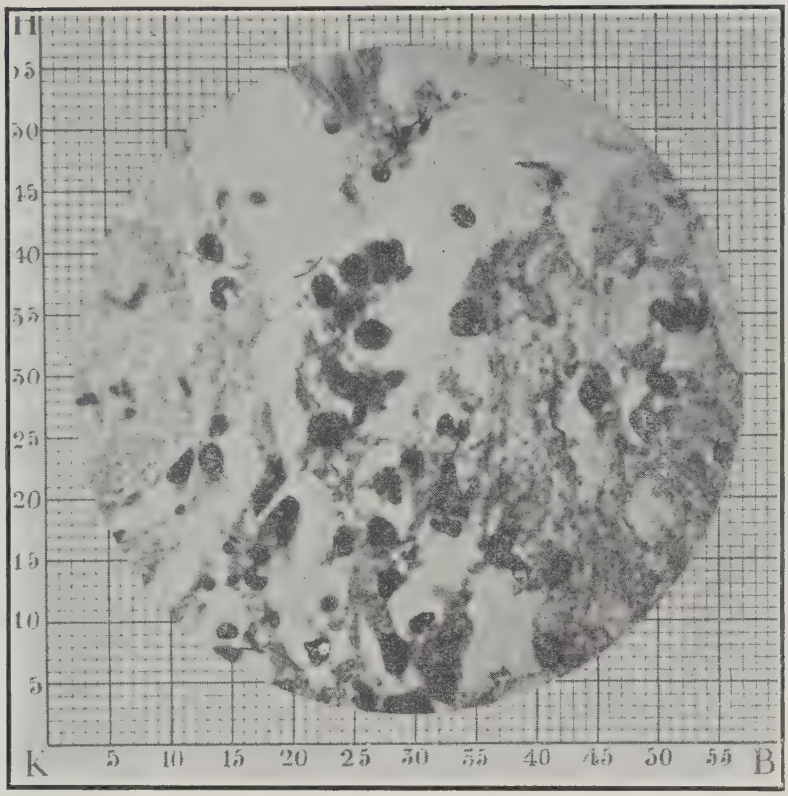


Fig. 94— x 1500. Chronic Pericementitis (Pocket in Pericementum). Bacteria in an ulcer at the border of a pericemental pocket.

H K 32 x K B 23, Diplococci.
H K 20 x K B 37, Bacilli.



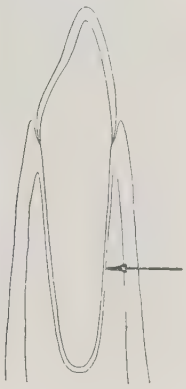


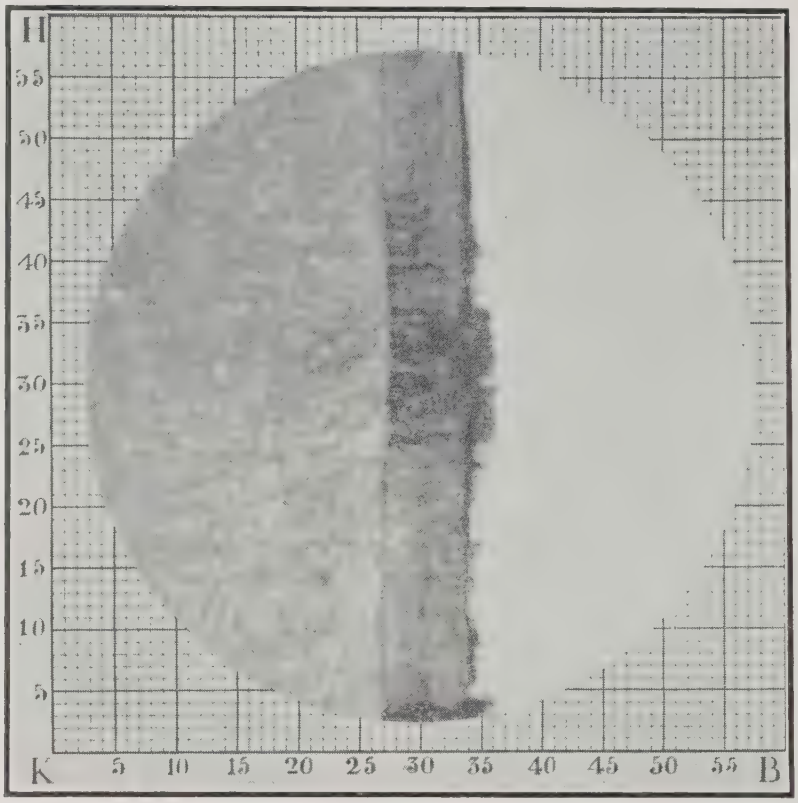
Fig. 95— x 1500. Chronic Pericementitis (Pocket in Pericementum). Bacteria in an ulcer at the border of a pericemental pocket.

H K 39 x K B 22, Bacilli.
Note occurrence of bacilli and cocci throughout the field.

ends of the principal fibres in the insertion layer, and occasionally to small remnants of necrotic pericementum adhering to the cemental surface. There is no miscroscopic evidence to show that in the average case the cementum is penetrated deeply by bacteria. That such penetration may take place is, however, shown by the accompanying photomicrographs (figs. 96 and 97).

We see no reason from the biological standpoint to agree with Black's statements regarding the impossibility of securing reattachment of the overlying soft tissues to denuded cementum. Black has laid down a postulate which may be summed up in the following sentence. "The key to the treatment of suppurative pericementitis is in the statement that suppurative detachments of the peridental membrane are permanent detachments."[2] On the contrary, clinical evidence has been accumulating for years to support the contention that reattachment is possible. We are now in a position to bring forth histological evidence which supports the claims of prominent clinicians and controverts the doctrine advanced by Black. The accompanying photomicrographs were taken from sections of a tooth treated by McCall. The case was shown as a clinic on reattachment at a meeting of the Alumni Association of the University of Buffalo in 1912. The claims of reattachment made to those who

[2]Special Dental Pathology, G. V. Black, p. 185.



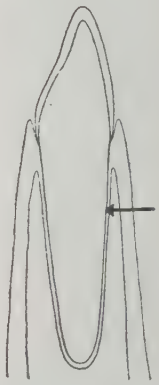
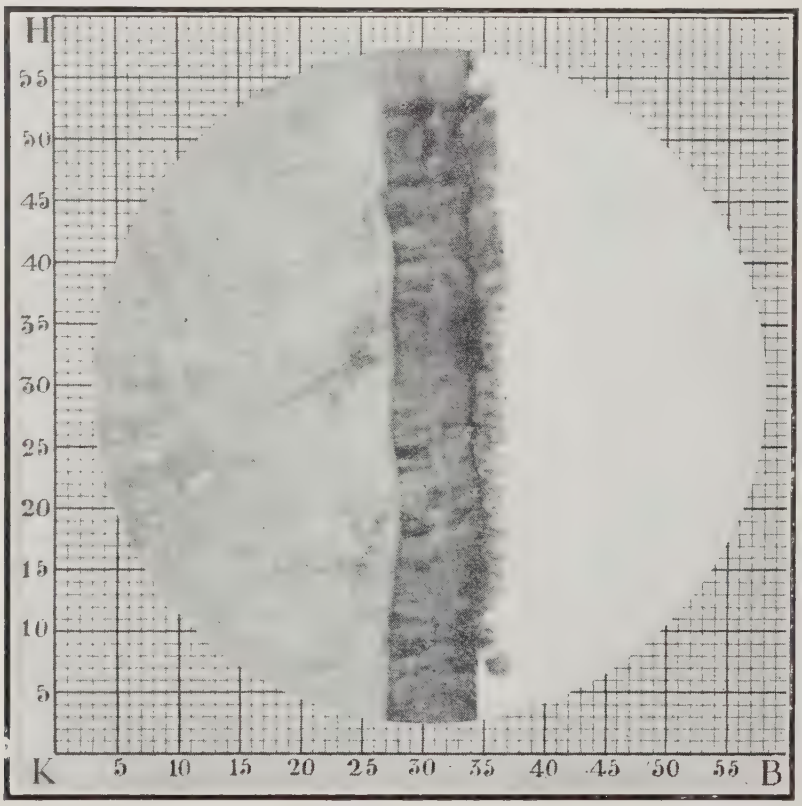


Fig. 96— x 250. Penetration of cementum by bacteria. This condition is comparatively rare. H K 15 x K B 31, Normal cementum.
H K 30 x K B 32, Cementum penetrated by bacteria.
H K 33 x K B 35, Bacteria on surface of cementum.



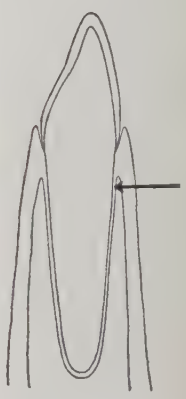
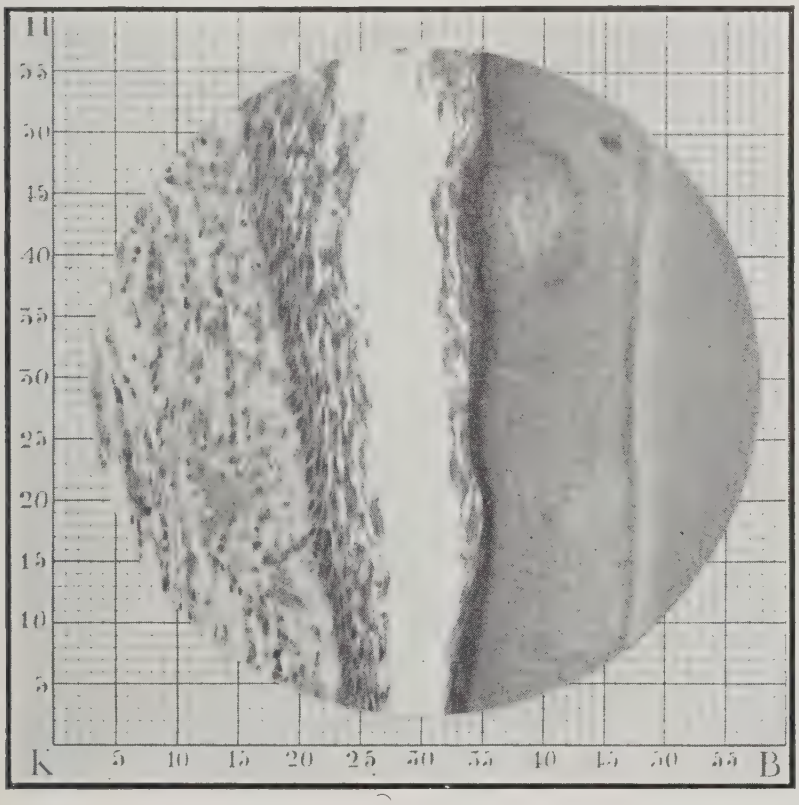


Fig. 97— x 250. Penetration of cementum by bacteria. Note that early penetration of the dentinal tubules by bacteria is shown.
H K 37 x K B 36, Bacteria on surface of cementum.
H K 44 x K B 28, Deep penetration of cementum by bacteria.
H K 33 x K B 25, Penetration of dentinal tubules by bacteria.

inspected the case were not questioned. This tooth and surrounding tissue was surgically removed in 1920. These sections show a reattachment of the connective tissue to the cementum surface. In addition, there is to be observed the phenomenon of a tenacious adherence of the crevicular epithelium to the the cementum. It will be noted that the connective-tissue fibres run parallel with the curetted cemental surface; also that the principal fibres, cementoblasts and epithelial cords typical of the normal are absent (figs. 98, 99, 100 and 101).

Not only has it been found that reattachment of the surrounding soft tissue to the cementum may be induced, but evidence is not lacking that regeneration of alveolar bone may take place. The supposed hopelessness of the building up of bone can readily be demonstrated to be without foundation, in fact, on the contrary many periodontists have offered radiographs which furnish indubitable proof of bone regeneration. The accompanying radiographs (figs. 102 and 103) are taken from the author's files. It will be noted in this case that the radiographs give evidence of a complete restoration of the alveolar crest, a type of bone regeneration usually regarded as least likely to be accomplished.



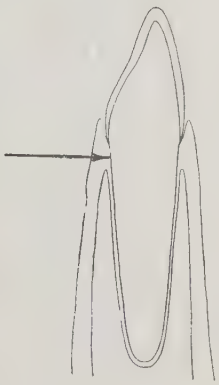
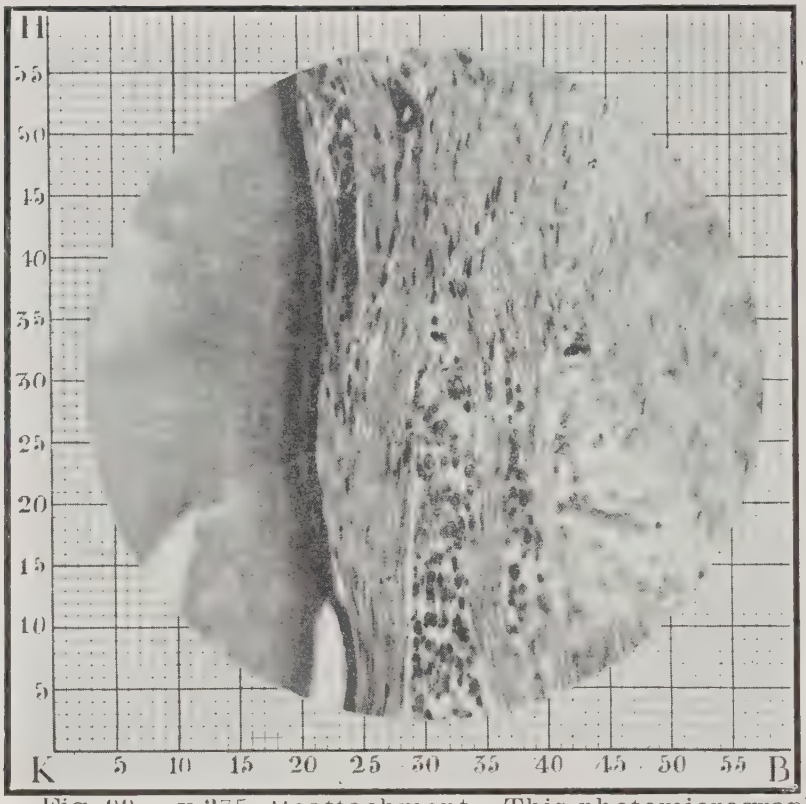


Fig. 98— x 375. Reattachment. This photomicrograph is taken near the top of the former pocket, and shows the tenacity of the adherence of the epithelium to the once denuded cementum. Note the wavy outline of the curetted cemental surface.

H K 35 x K B 35, Curetted cemental surface.
H K 35 x K B 32, Adherent epithelium.
H K 35 x K B 29, Space produced by tearing of epithelium.



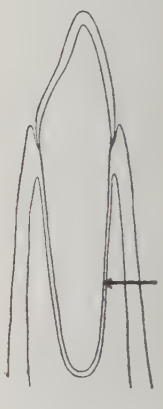
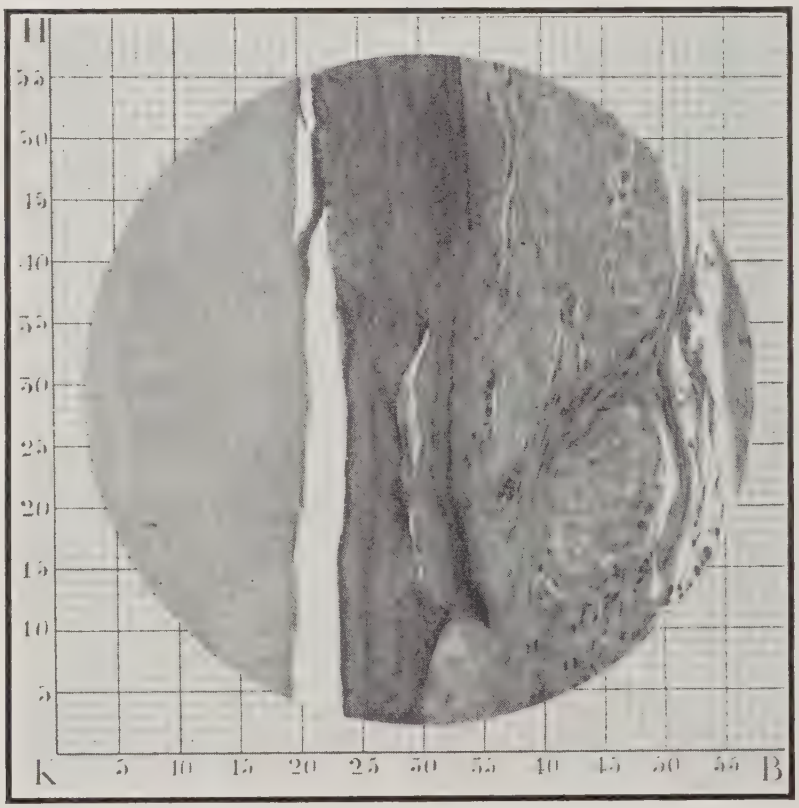


Fig. 99— x 375. Reattachment. This photomicrograph shows reattachment of the connective tissue farther down in the former pocket. Note that the connective-tissue fibres lie parallel with the cemental surface. Note also absence of principal fibres, cementoblasts and epithelial cords.

H K 35 x K B 22, **Cemental surface.**
H K 18 x K B 27, Connective-tissue fibres.
H K 44 x K B 24, Blood-vessel.



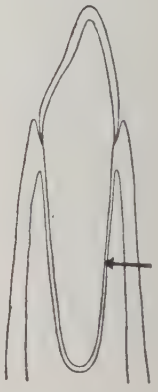


Fig. 100. Reattachment. New cementum laid down over the curetted cemental surface. While certain areas show this condition, yet the chief feature of the process is the reattachment of connective tissue to the original curetted surface.

H K 49 x K B 35 }
H K 25 x K B 33 } Secondary cementum laid down over curetted cementum.

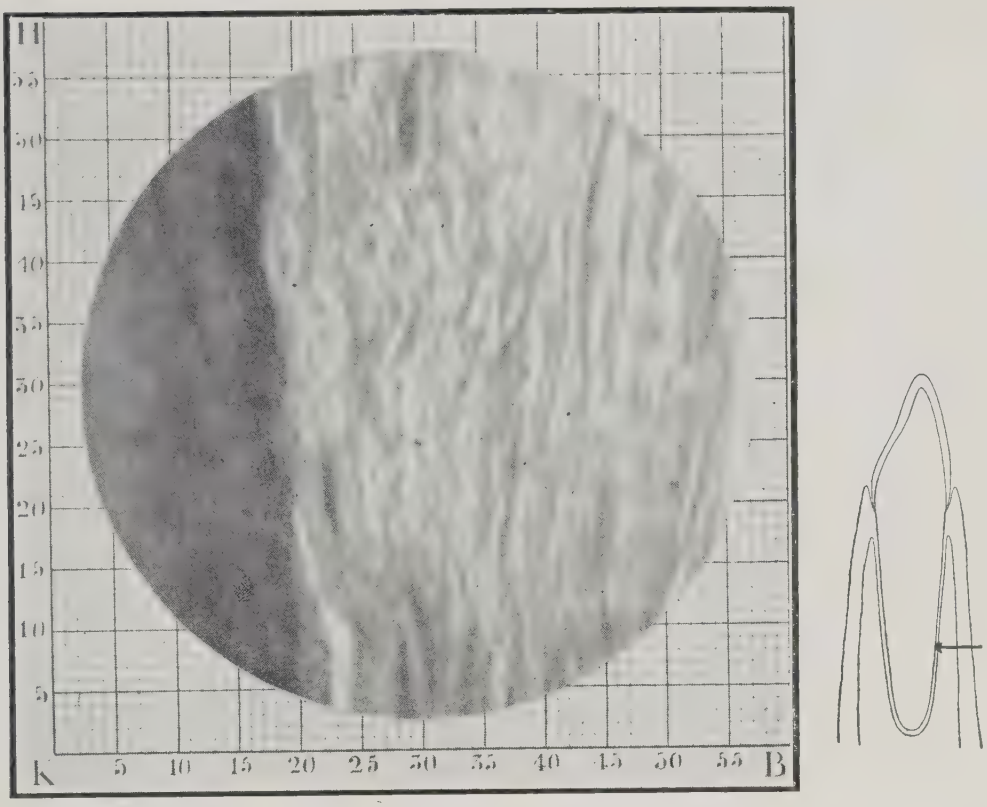


Fig. 101— x 2250. Reattachment. This photomicrograph shows the connective-tissue cells and fibres lying parallel to the cemental surface.

H K 52 x K B 29
H K 30 x K B 33 } Connective-tissue cells.
H K 5 x K B 28

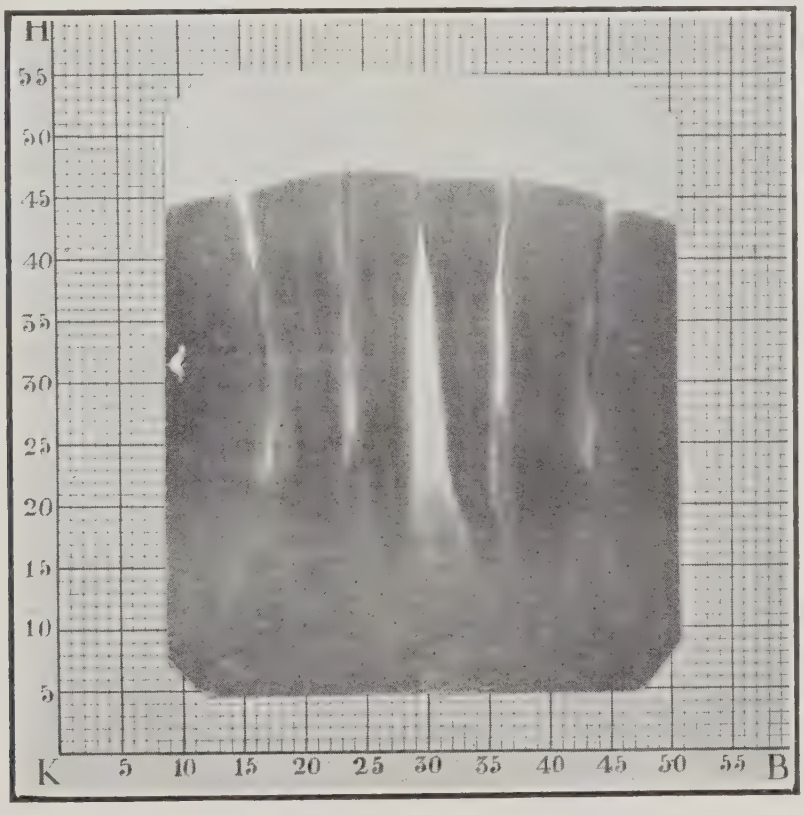


Fig. 102—Regeneration of Alveolar Bone. Radiograph taken Mar. 1, 1921. Note that marked rarefaction of bone has occurred in the septal bone between lower central (median line). Age 43.

Fig. 103—Regeneration of Alveolar Bone. Radiograph taken Mar. 1, 1922, at approximately the same angle as the preceding radiograph. Note the height of the restored alveolar crest as compared with the height of the alveolar crests between the adjoining teeth. Note formation of an apparently normal lamina dura.

In view of the evidence just submitted, it is interesting to refer to the pessimism of most writers on this subject, regarding the possibility of cure in a mature case of Chronic Periodontitis, Complex. That this pessimistic attitude has not been justified may be demonstrated through a review of the following fundamental postulates whose truth we believe has been established in this paper.

1. The progressive nature of both of the basic lesions (Rarefyfying Pericementitis Fibrosa and Infective Periodontitis). It will be recalled that the regressive changes in the alveolar bone are secondary to progressive changes in the pericementum.
2. The demonstration that reattachment of the soft tissues to the cementum may be induced.
3. The demonstration that rarefaction of the alveolar bone can be arrested, and that, in many cases, an actual regeneration of the alveolar bone may be induced.

These three postulates may be summed up in the form of the following proven hypothesis,—*Chronic Periodontitis belongs in the category of curable diseases.*

Deposits of calculus are not, as a rule, found extending to the depth of the pocket. Almost invariably, in pocket formation, the soft tissues are detached from the cemental surface for a considerable distance in advance of the deposition of calculus. Serumal calculus is, therefore, not a primary agent in pocket extension. Its precipitation occurs subsequent to the separation of the soft tissues from the cementum. These concretions act as mechanical injurious agents, and also as a source of chemical irritation, e.g., toxins derived from contained bacteria. They thus are to be looked upon as sustaining factors in the maintenance of inflammation of the overlying soft tissue (figs. 104 and 105).

The type of exudation from periodontal pockets is commonly known as pus, and is make up of leukocytes in abundance, serum in varying quantities, fibrin usually of scant amount, and living and dead bacteria. Of the leukocytes, the polymorphonuclear predominate although endothelial leukocytes and lymphocytes may be fairly abundant.

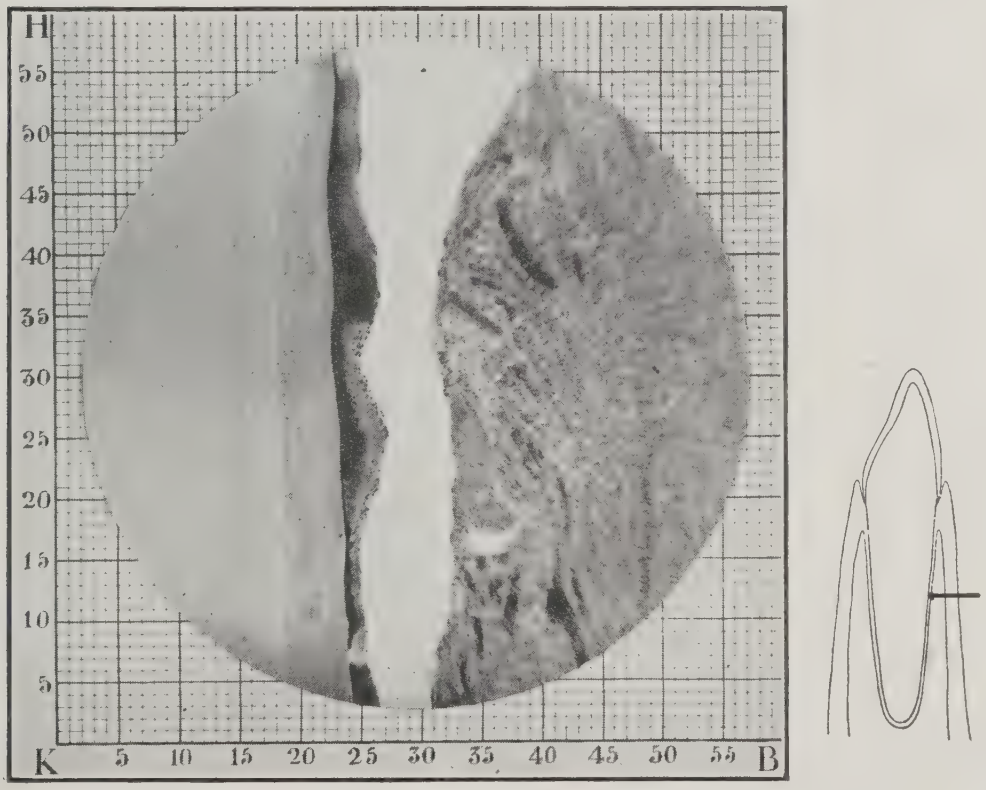


Fig. 104— x 125. Calculus on cemental surface in pocket.

H K 30 x K B 21, Cementum.
H K 38 x K B 25, Calculus.
H K 30 x K B 30, Pocket.
H K 30 x K B 40, Soft tissue.
H K 40 x K B 39, Engorged blood-vessel.

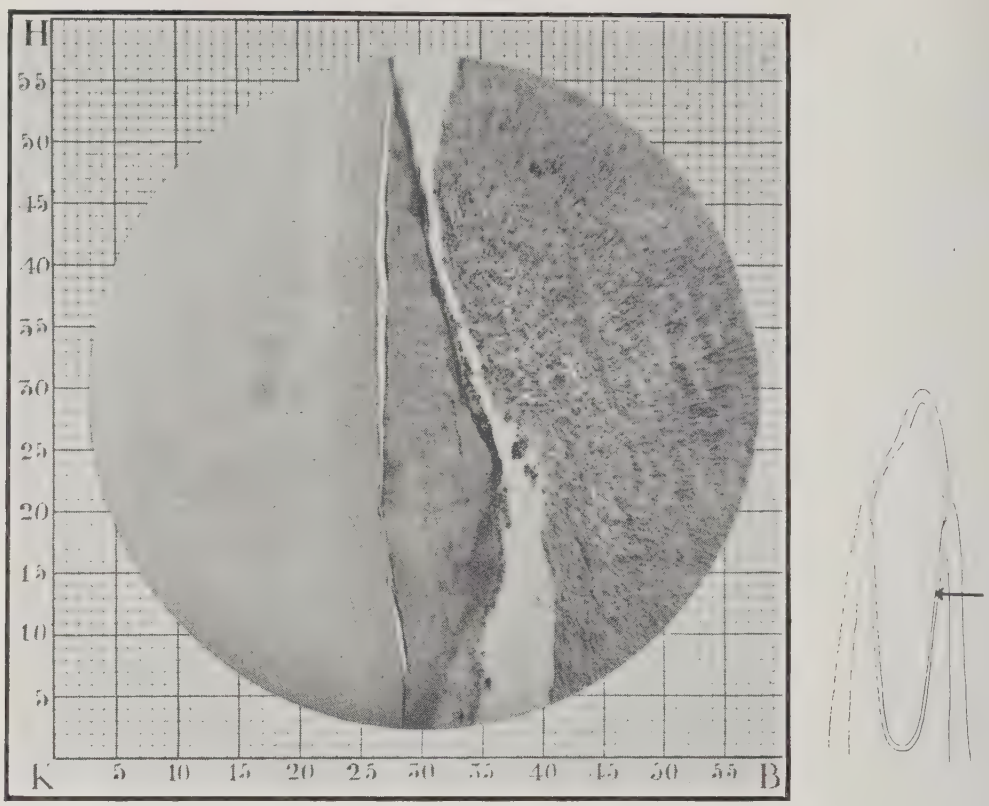


Fig. 105— x 125. Calculus on cemental surface in pocket.

H K 25 x K B 32, Calculus.
H K 25 x K B 45, Soft tissue.
Note that entire surface of soft tissue is covered with epithelium.

PHASE 3. INFECTIVE OSTEITIS (RAREFYING).

It has been stated that alveolar resorption has taken place, without infection and in advance of the extension of the pocket. The deepening pocket on account of its sheltered location and its contained débris, serves as an incubator which is particularly favorable to the growth of micro-organisms. Through increased absorption of toxins, and actual infection, the phenomena of infective osteitis are added to those of non-infective rarefaction of the alveolar bone. In other words, the inflammatory process brought about by infection extends to the alveolar bone, and through its contributing factors, e.g., acids produced by bacterial action on the fluids of the environment, products arising from cell destruction, and secretory products of abnormal tissue-cells, further absorption takes place.

In the fibrillar connective tissue which has been formed subsequent to the non-infective absorption of bone, there is to be noted an extensive small-cell infiltration, which also, in certain areas, has entered the marrow-spaces. The activity produced by chronic inflammation in the soft tissues in the marrow-spaces or Haversian canals results in certain striking changes in the dense basement-substance of the bone. As a result of these changes, either the calcified ground-substance is absorbed and replaced by young cellular forms of fibrillar

connective tissue or the marrow-spaces undergo progressive reduction in size by new-formed lamellae on their walls. These alterations are named, respectively, rarefying and condensing osteitis, which terms simply indicate the local results of inflammation.

The rarefaction of the bone in infective rarefying osteitis occurs by enlargement of the medullary spaces and Haversian canals, with consequent disappearance of the spongy bone substance. The soft structures of the bone become very cellular and vascular and are converted into granulation tissue. Throughout this tissue, there is a marked infiltration of lymphocytes and plasma cells. Under the influence of this tissue the ground-substance of the bone becomes absorbed. (Figs. 106, 107, 108, 109, 110 and 111.)

A prominent feature of the histological picture is the marked disturbance of pericemental circulation. The blood-vessels are greatly dilated, and, in many instances, the red blood-cells have escaped into the surrounding tissues. Such hemorrhages are usually small, but their occurrence, on the other hand, may be wide-spread throughout the pericementum. (Fig. 112.)

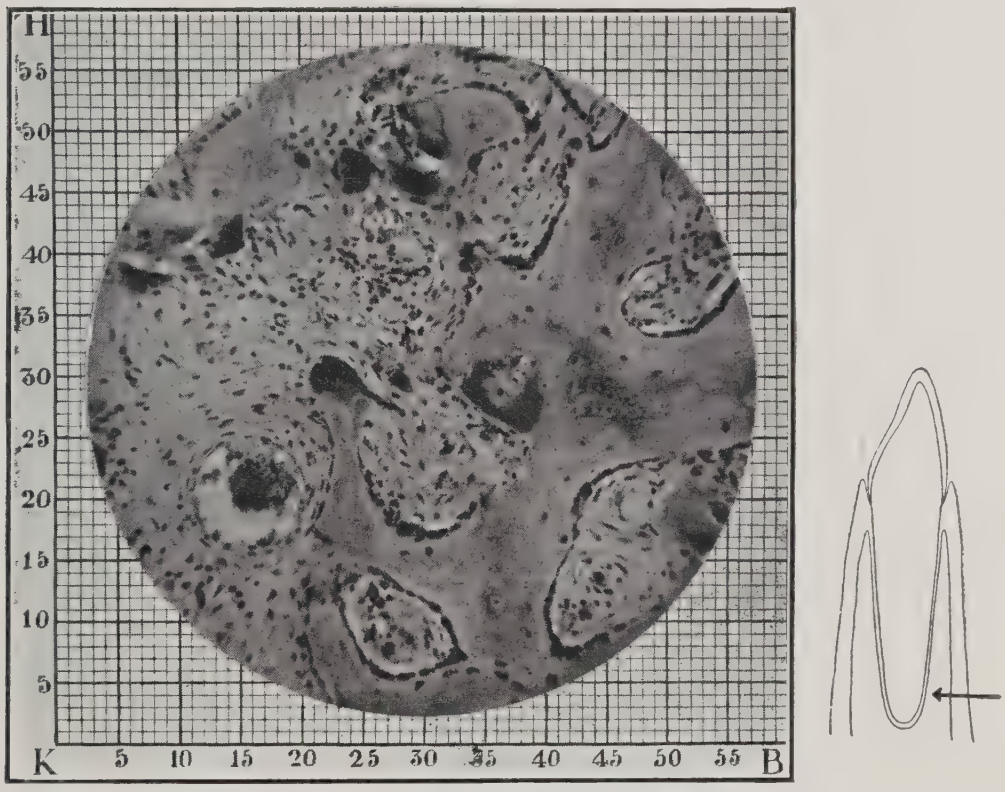
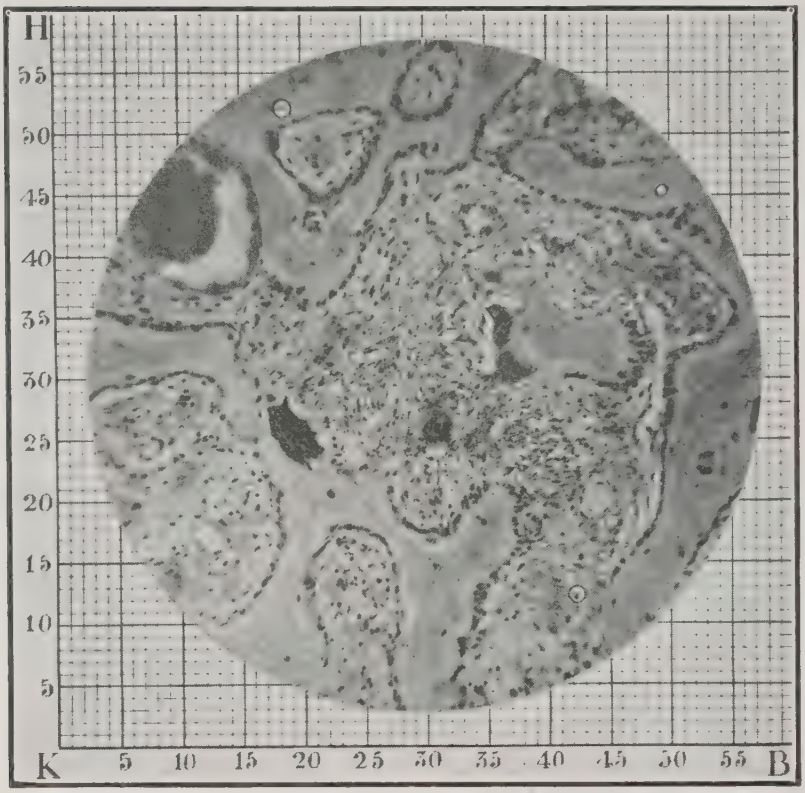


Fig. 106. Infective Osteitis (Rarefying). This photomicrograph shows trabeculæ of alveolar bone undergoing absorption. Note the leukocytic infiltration of the soft tissue in the medullary spaces.

H K 20 x K B 25, Trabecula of alveolar bone.
H K 30 x K B 22, Osteoclast absorbing alveolar bone.
H K 17 x K B 29, Alveolar bony trabecula undergoing absorption.
H K 39 x K B 27, Leukocytic infiltration.
H K 21 x K B 17, Engorged blood-vessel.



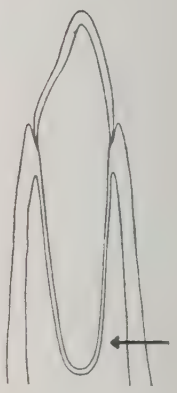
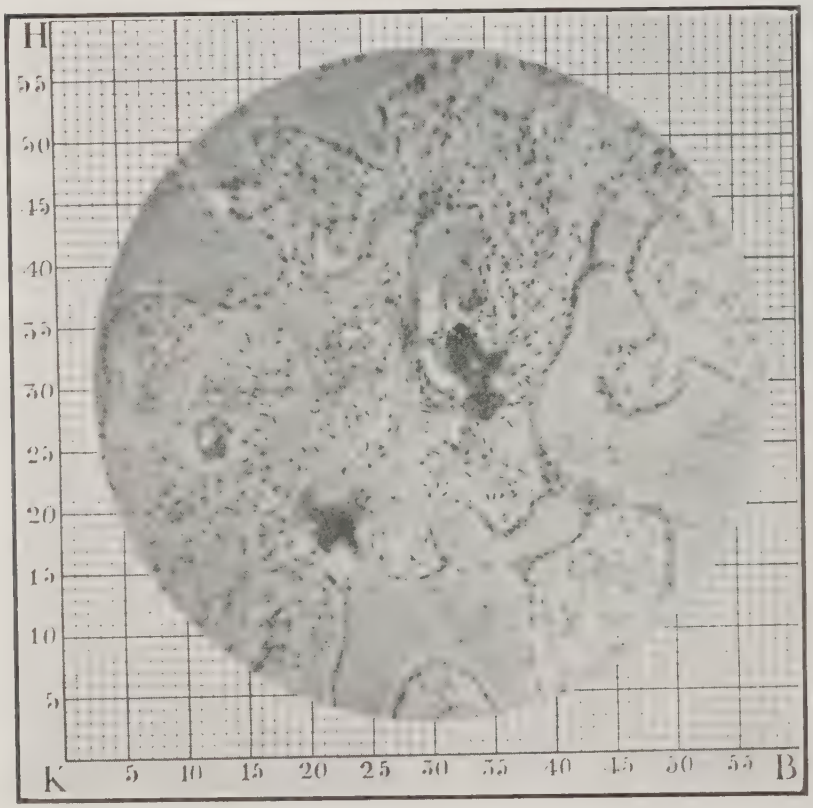


Fig. 107. Infective Osteitis (Rarefying).
H K 26 x K B 20, Osteoclast. H K 26 x K B 36, Leukocytic infiltration.
H K 34 x K B 41, Remnant of bony trabecula.



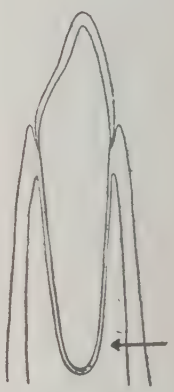
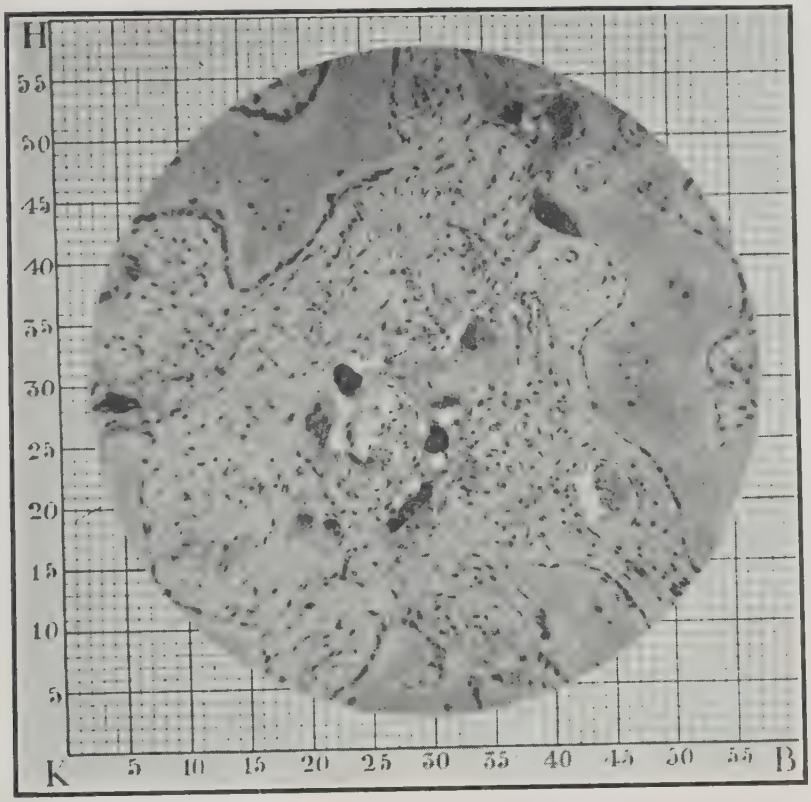


Fig. 108. Infective Osteitis (Rarefying).
H K 19 x K B 22 } Osteoclasts.
H K 31 x K B 34 }
H K 22 x K B 40, Osteoblasts. Note the continuous line of osteoblasts on the surface of the bony trabeculae.
H K 40 x K B 37, Leukocytic infiltration.



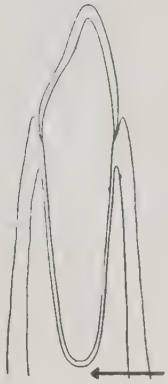


Fig. 109. Infective Osteitis (Rarefying).
H K 31 x K B 23 Osteoclasts.
H K 44 x K B 40
H K 31 x K B 33, Remnant of bony trabecula.
H K 27 x K B 28, Leukocytic infiltration.
H K 21 x K B 45, Engorged blood-vessel.

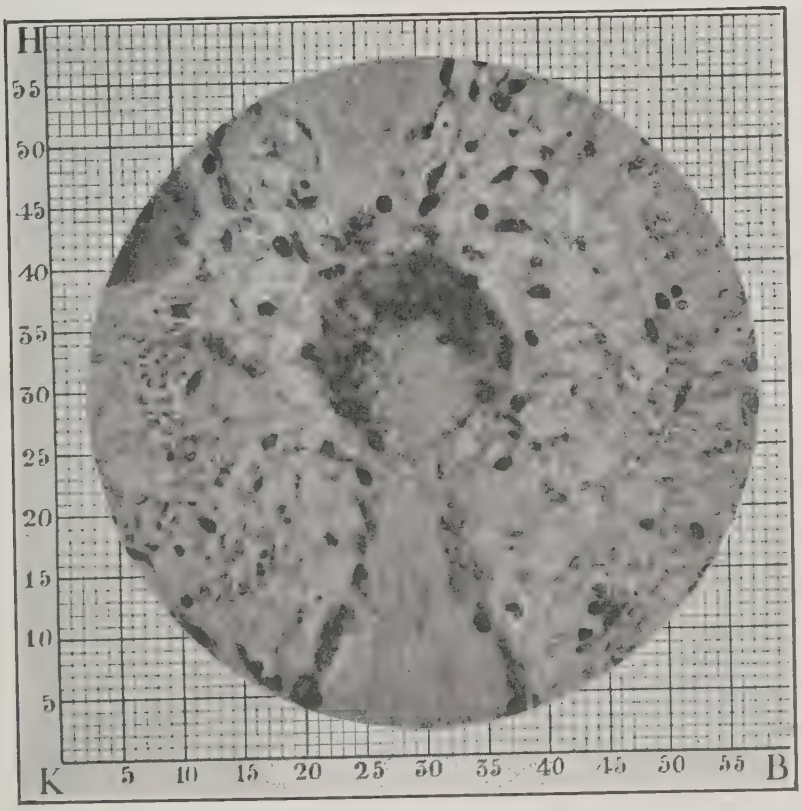


Fig. 110— x 750. High magnification of an osteoclast at the extremity of a bony trabecula.
H K 30 x K B 30, Extremity of bony trabecula.
H K 38 x K B 30, Crescentic osteoclast.

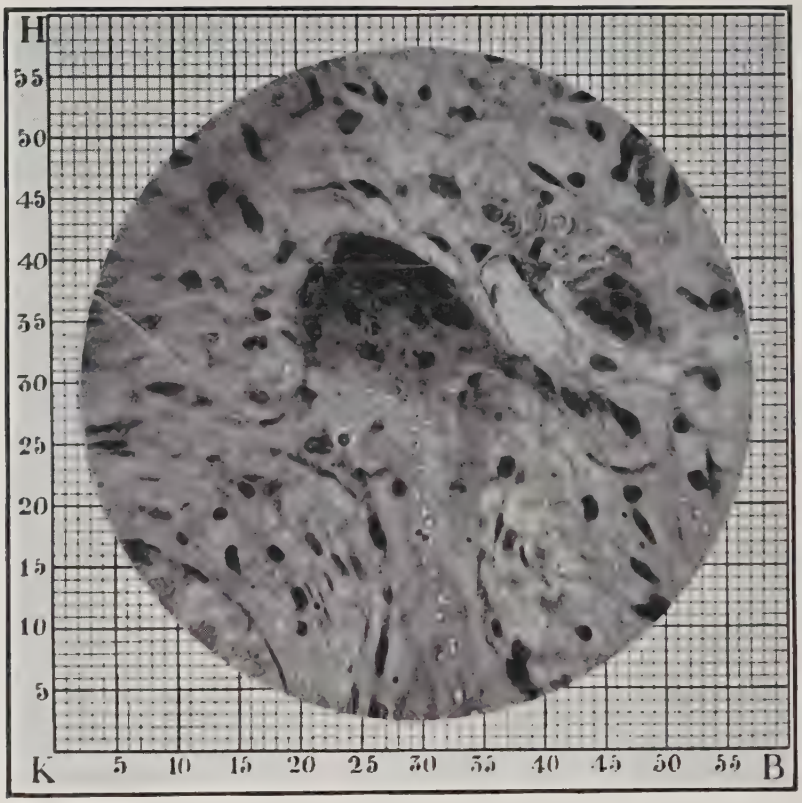


Fig. 111— x 750. High magnification of an osteoclast.
H K 28 x K B 30, Extremity of bony trabecula.
H K 31 x K B 27, Cytoplasm of osteoclast.
H K 40 x K B 27, Multiple nuclei of osteoclast.

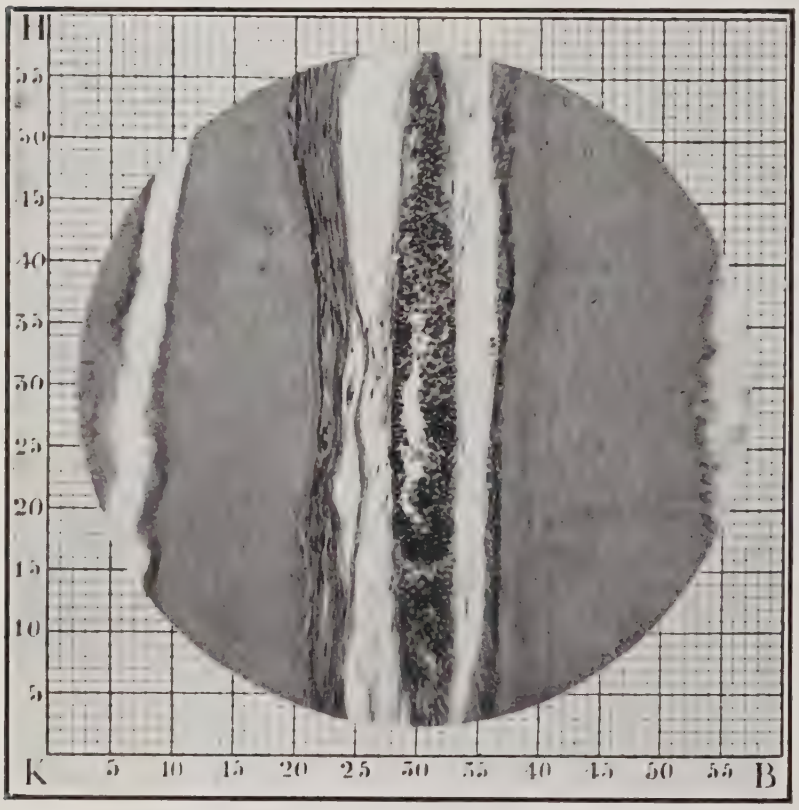


Fig. 112— x 250. Dilatation of a blood-vessel in the pericementum in Infective Osteitis.
H K 30 x K B 31, Blood-vessel.
H K 30 x K B 22, Border of cementum.
H K 30 x K B 36, Border of alveolar process.

In condensing osteitis, through increased osteoblastic activity, the lamellae undergo progressive thickening and the marrow-spaces and Haversian canals are correspondingly reduced. In the alveolar process, this condition is frequently noted about teeth in over-function. Here it may be looked upon as a compensatory effort on the part of the bone to maintain a state of mechanical equilibrium. The toxins of bacteria of low virulence, by their mild irritation, are also regarded as causative factors in the production of condensing osteitis. (Figs 113 and 114.)

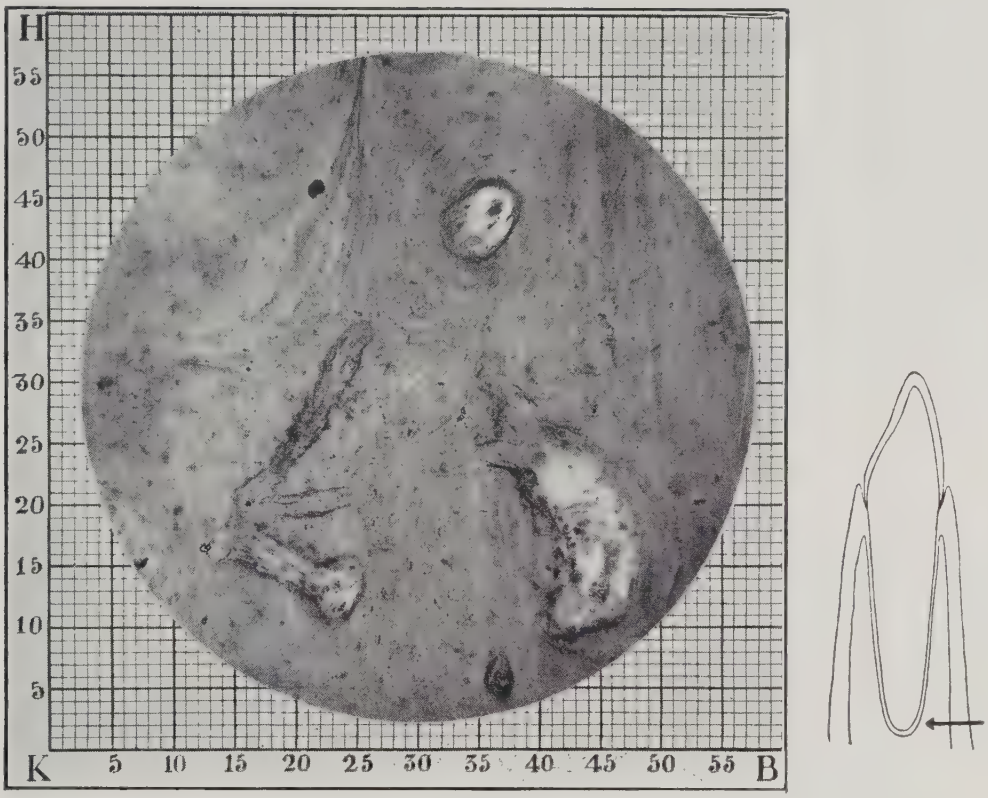


Fig. 113— x 187. Condensing Osteitis.

H K 28 x K B 22 } Medullary spaces obliterated by new bone formation.
H K 20 x K B 21 }
H K 26 x K B 39, Medullary space in process of obliteration.

A modification of Chronic Periodontitis, Complex may occur when when this disease condition involves the tissues in the bifurcation of the roots of any multi-rooted tooth.

The lesions occur in the following order:

1. Rarefying Pericementitis Fibrosa, as described previously.
2. Chronic Gingivitis (cemental-gingival pocket).
3. Chronic Pericementitis (pericemental pocket).
4. Formation of granuloma in the bifurcation. The soft tissue remaining in the bifurcation after the alveolar rarefaction, is converted into a granuloma by the invasion of bacteria from the pocket (figs. 115, 116, 117, 118).
5. Infective Osteitis (Rarefying).

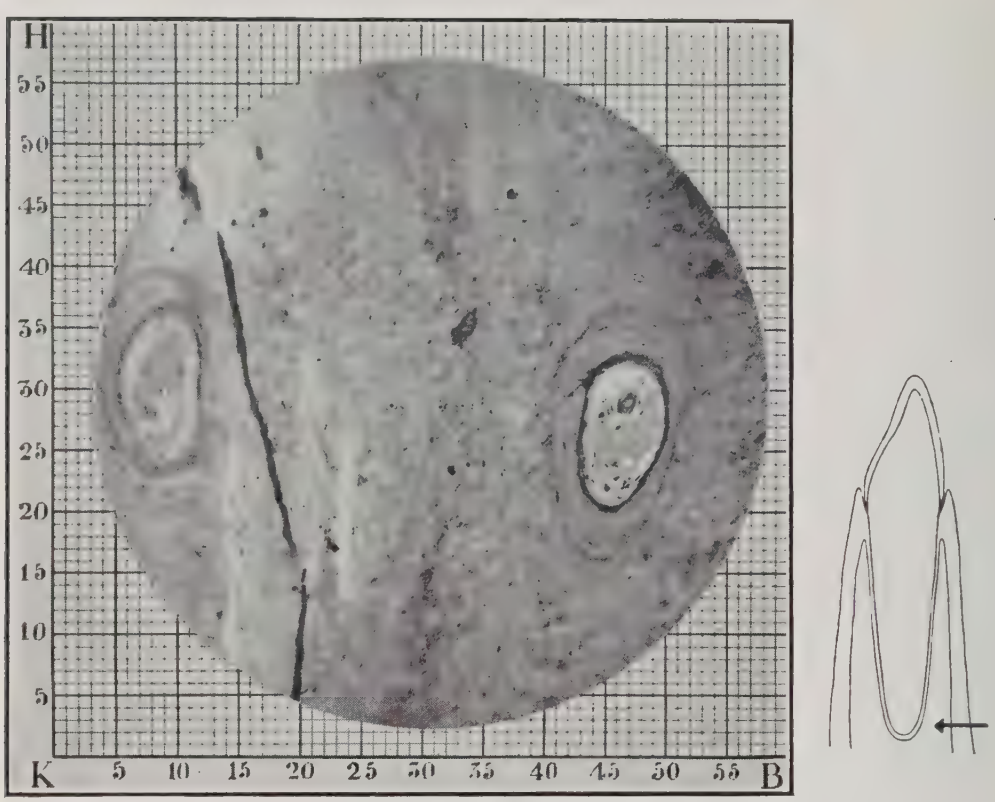


Fig. 114— x 187. Condensing Osteitis.

H K 38 x K B 8 } New lamellae of bone being laid down around medul-
H K 35 x K B 46 } lary spaces.

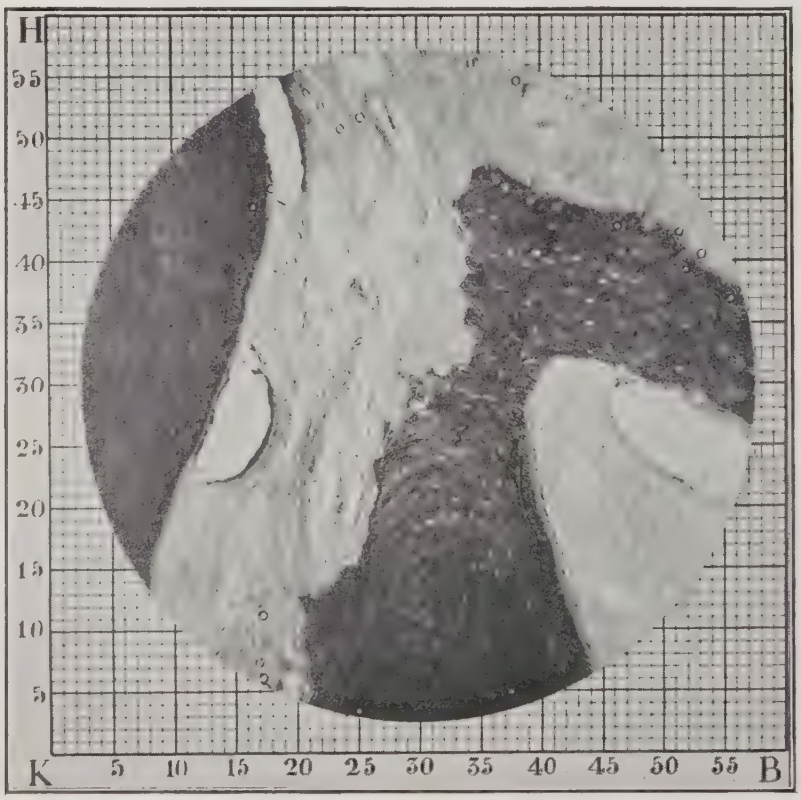


Fig. 115— x 94. Rarefying Pericementitis Fibrosa. Rarefaction of the alveolar crest in the bifurcation of a lower molar.

H K 33 x K B 34, Excavations in the border of alveolar bone.
H K 35 x K B 27, Pericementum.
H K 40 x K B 15, Cementum.
H K 30 x K B 42, Absorption in the body of the alveolar bone.

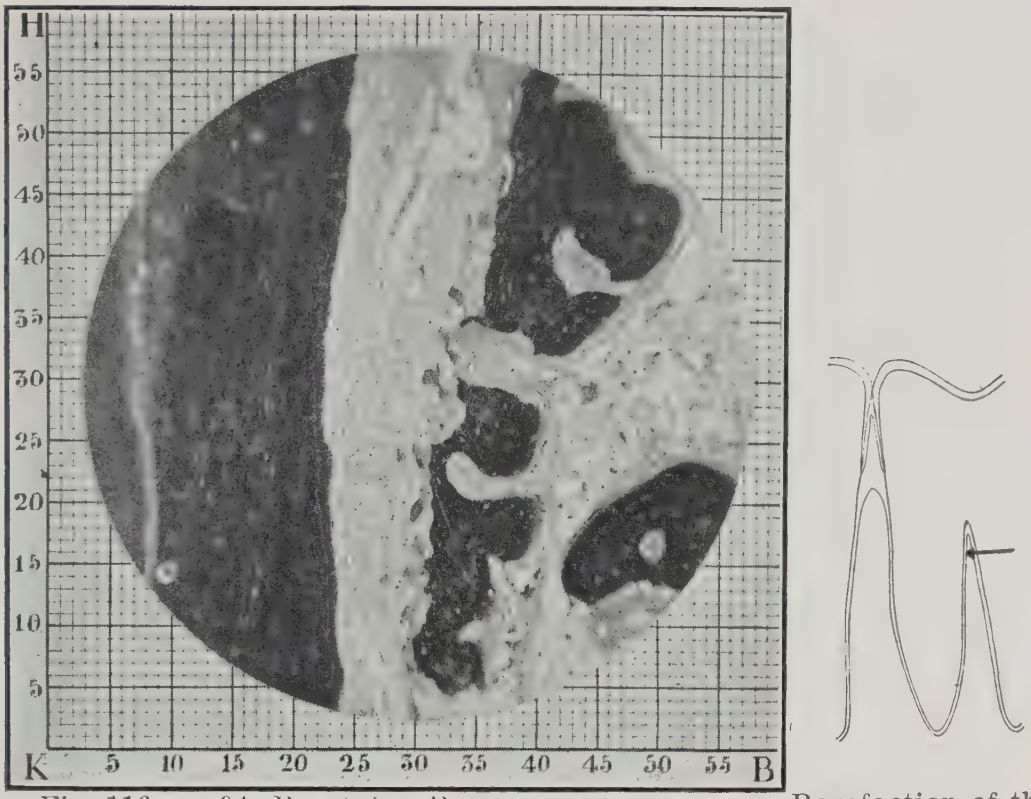


Fig. 116— x 94. Rarefying Pericementitis Fibrosa. Rarefaction of the alveolar process in the same specimen as shown in the preceding illustration. The alveolar bone is undergoing segmentation.

H K 25 x K B 28, Pericementum.
H K 25 x K B 23, Border of cementum.
H K 40 x K B 40 } Segments of alveolar bone.
H K 16 x K B 46

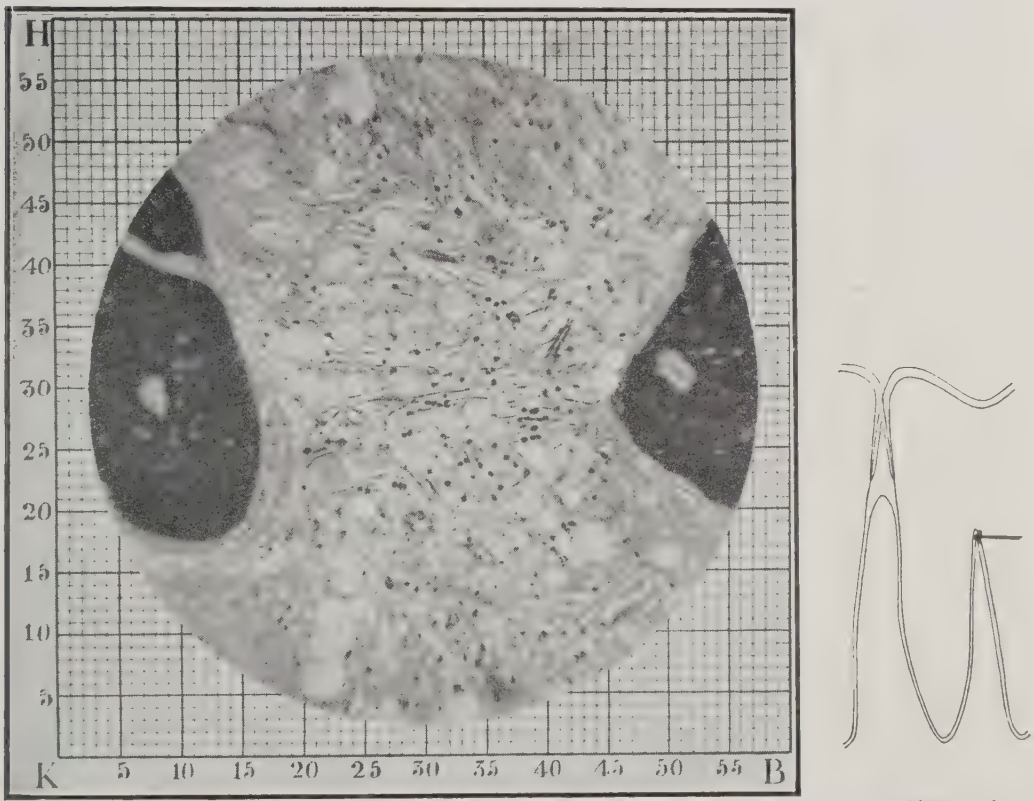


Fig. 117— x 125. Leukocytic infiltration in the connective tissue which has replaced the alveolar bone in the bifurcation. Beginning of granuloma formation.

H K 28 x K B 39, Leukocytic infiltration.
H K 28 x K B 13 } Remnants of alveolar bone.
H K 25 x K B 53 }

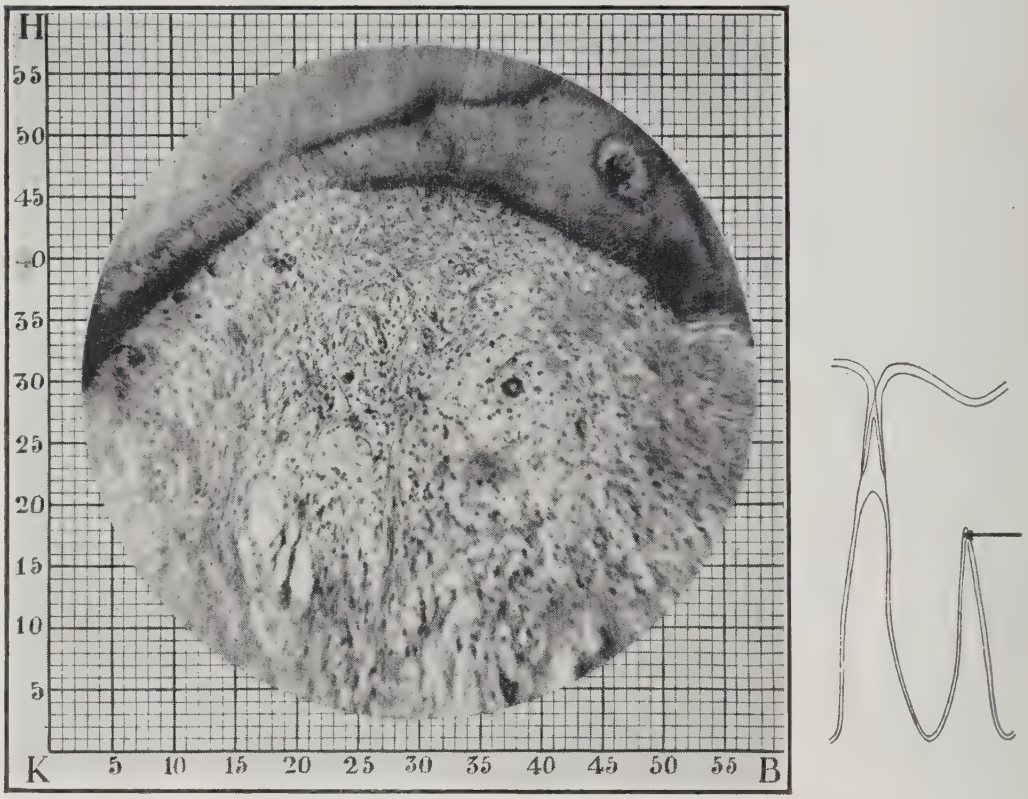


Fig. 118— x 125. Granuloma in bifurcation.
H K 50 x K B 23, Dentinal-cemental junction.

PART V:

HISTO-PATHOLOGY OF THE PERIAPICAL TISSUES.

Periapical diseases contiguous to the cementum, and with which the practitioner comes into contact daily, may be divided into three main classes:

(1) Granulomata
(2) Abscesses
(3) Dental Cysts.

In general, a granuloma represents a reaction to a mildly injurious agent, exemplified in the periapical tissues by bacteria low in virulence, chemicals in low concentration used in root canal treatment, and mechanical agents not severe in character. The actual injury by these types of injurious agents is slight, as a rule, and seldom can be shown morphologically. On the other hand, the inflammatory reaction is usually marked and frequently leads to serious consequences. This reaction, as demonstrated in the dental granuloma, always takes place at the site of injury, the usual location being the apex of a tooth. However accidental perforation of the bifurcation of a multi-rooted tooth, or through the cementum on the side of the tooth, quite often leads to the production of this lesion in these areas.

1. GRANULOMATA.

A granuloma consists, as the name implies, chiefly of granulation tissue. At the expense of the resorption of the alveolar lamina dura, the granuloma increases in size, and finger-like extensions project into the medullary spaces of the bone. During the essayist's work on the subject, Thoma's classification was studied, and will be used in the following.

(*a*) *Simple.*

This type of granulomata consists chiefly of a proliferation of vascular endothelium and fibroblasts. The site of the injury becomes infiltrated with lymphocytes and, in smaller numbers, polymorphonuclear leukocytes. Endothelial cells are frequently seen in large numbers, owing to the presence of certain foreign substances. A conspicuous feature of nearly all granulomata is the presence of many plasma cells, easily differentiated from the lymphocytes by their eccentric nuclei, often two to four in number. They also differ from the lymphocytes in the "large increase in the amount of cytoplasm, which has marked basophilic properties, except for a small acidophilic area adjoining the nucleus, in which the centrosomes are always found." (Mallory.)

The function of the lymphocytes is believed to be the neutralization and disposal of injurious substances absorbed through the lymphatics. With increase in size of the granuloma and absorption of the lamina dura, its outer portion is characterized by the arrangement in bundles of connective-tissue fibres, which lie, as a rule, in a direction parallel with its outer surface. This has been termed the capsule. Extensions of the capsule project into the medullary spaces of the bone. Examination of a number of sections very often reveals the presence of small areas of osseous tissue, which has become isolated from the alveolar process. The capsule is highly vascularized, numerous fairly large blood-vessels maintaining communication between the inner parts of the granuloma and the surrounding bone (figs. 119, 120 and 121).

Beneath the capsule, there may be distinguished a much thicker layer of less dense reticular connnective tissue, containing a great number of lymphocytes and plasma cells, and some polymorphonuclear leukocytes.

(*b*) *Epitheliated.*

When the pericementum contains the epithelial cords described previously, "the masses epitheliaux of Malassez," and upon involvement of these cells in the inflammatory process, a striking feature of the microscopic picture is their proliferation through the tissue, in the form of plugs, strings or trellis-work. Winding and branching cords often constitute a net-work of epithelial cells through the lesion. This type is known as an epitheliated granuloma. (fig 122).

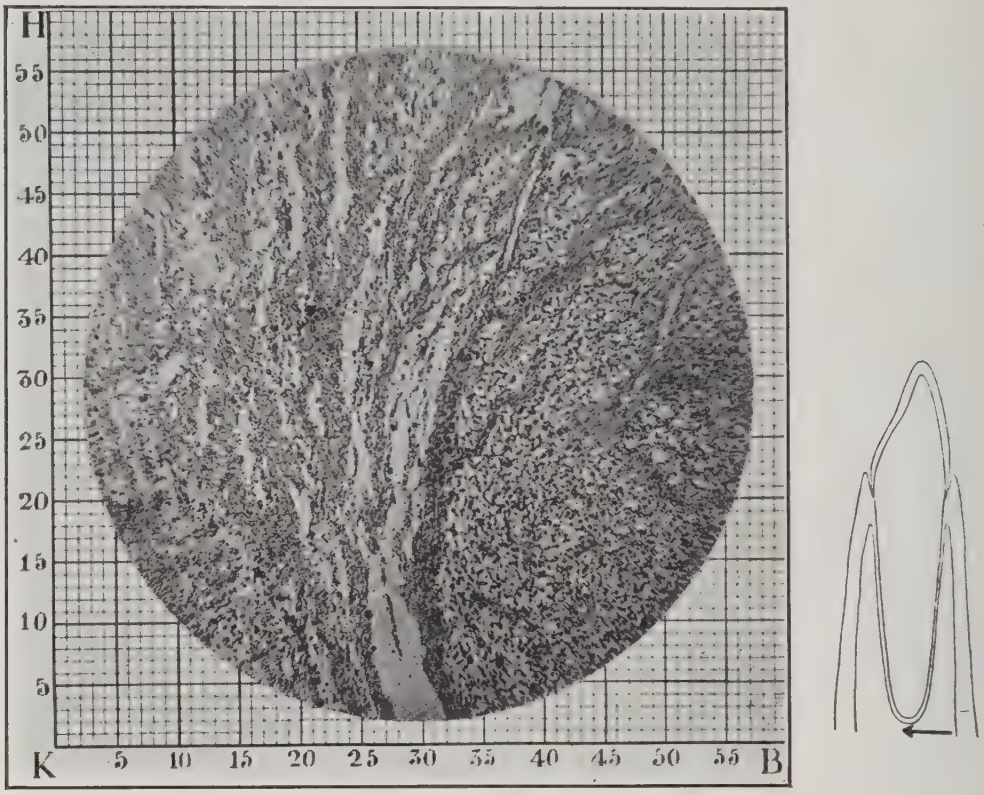


Fig. 119— x 94. Simple granuloma, showing connective-tissue capsule.
H K 25 x K B 32, Connective-tissue capsule.
H K 20 x K B 43, Granulation tissue.

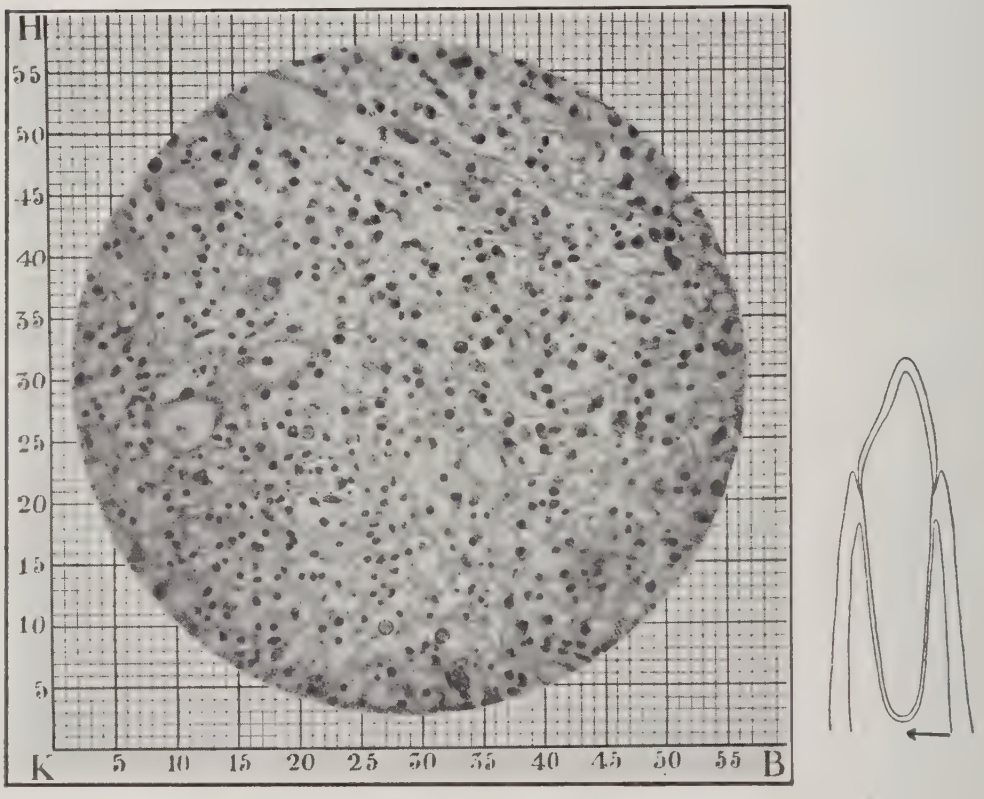
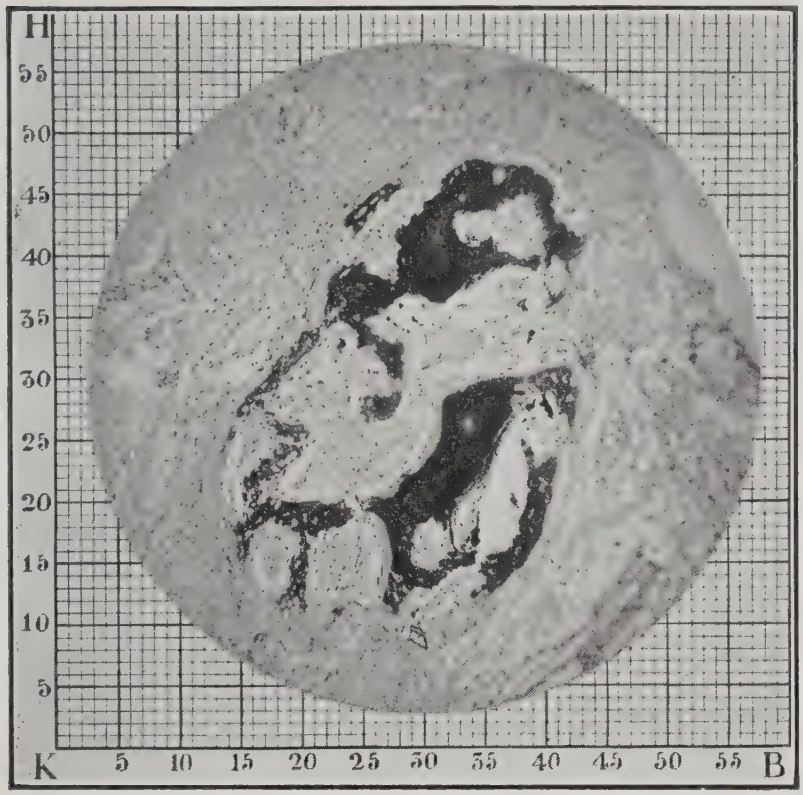


Fig. 120— x 375. Simple granuloma.
H K 50 x K B 29, Proliferating endothelial cells forming a new blood-vessel.
H K 39 x K B 26
H K 27 x K B 37
H K 27 x K B 27
} Plasma cells.



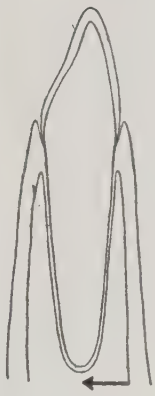


Fig. 121— x 94. Remnant of alveolar bone surrounded by granulation tissue and undergoing absorption.

H K 40 x K B 31
H K 23 x K B 33
} Large segments of alveolar bone.

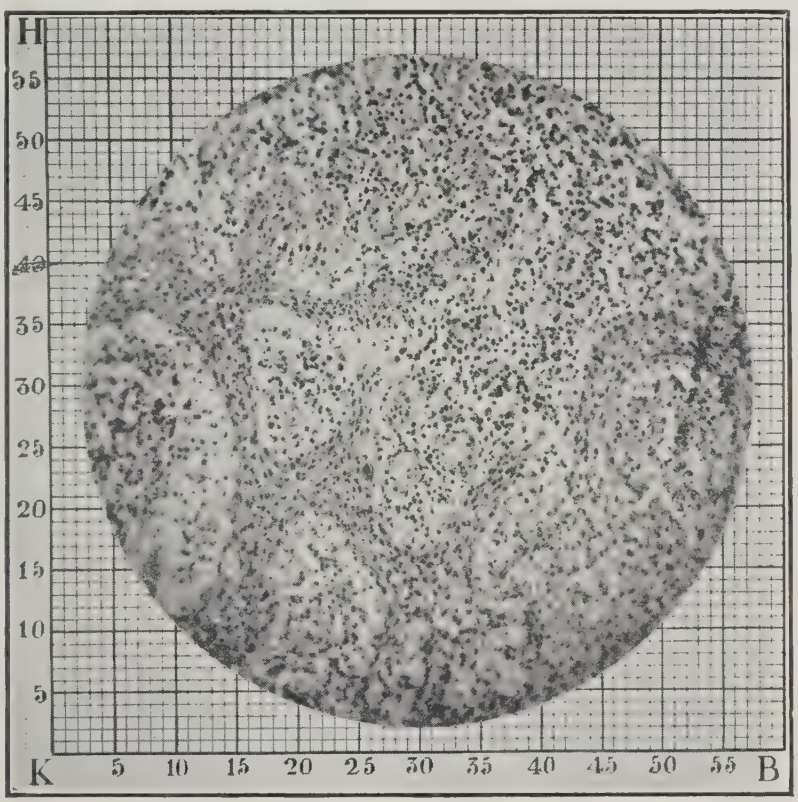


Fig. 122— x 188. Epitheliated granuloma.

H K 30 x K B 15
H K 21 x K B 35
H K 35 x K B 48
} Proliferating epithelium.

(*c*) *Granuloma showing Necrosis.*

Here and there throughout certain granulomata, areas may be noted where a number of cells are separated from the others and have undergone degeneration. Usually in this type there is an increase in the number of polymorphonuclear leukocytes. The essayist regards this lesion as a potential abscess. When the destructive process becomes extensive and suppuration results, if the connective-tissue capsule is not broken down, the condition is known as blind abscess. In certain cases, however, a sinus to the gum is produced.

(*d*) *Dental Granulomata showing Retrograde Processes.*

Evidence of cholestrin formation is seen in many sections of granulomata. Cholestrin crystals can be recognized by the acicular spaces left by the crystals during dehydration in alcohol (fig. 123).

Fig. 123— x 94. Granuloma showing cholesterin formation.

H K 30 x K B 25 } Spaces in the section formerly occupied by cholesterin
H K 30 x K B 34 } crystals.

(*e*) *Dental Granulomata showing Cyst Formation.*

In epitheliated granulomata, showing, in certain areas, evidence of degenerative processes, the epithelial cords, previously described, manifest a definite tendency to surround and encapsulate such foci. This can be looked upon as the beginning of root-cyst formation. True simple granulomata contain no epithelial cell clusters, and for this reason can never develop into these root-cysts. Dewey, in speaking of beginning cyst formation, has stated: "Strands grow out from

this epithelial layer into the granulation tissue and encircle and snare off other regions, which, being cut off from the surrounding tissues, are poorly nourished, and hence the tissue undergoes degenerative processes. When these are complete, the epithelial barrier is also dissolved and the detritus added to the cystic content" (fig. 124).

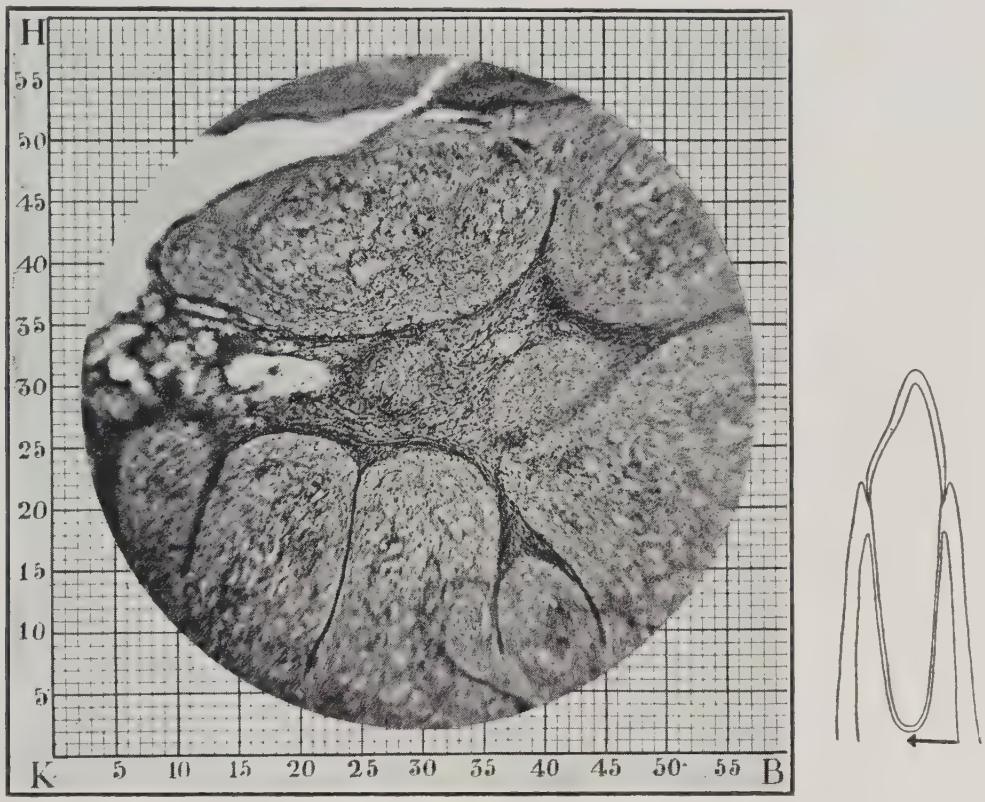


Fig. 124— x 9. Cystic granuloma.

H K 55 x K B 27, Apical cementum.
H K 20 x K B 12 } Proliferating epithelial strands tending to encircle
H K 15 x K B 23 } Adjacent tissue.
H K 18 x K B 39 }

On the same question, Prieswerk has written the following: "The epithelial cells generally lie close together, but in some areas they are interrupted by minute lacunae, which are filled with granular fragments of degenerated cells. Such lacunae may be considered primitive cysts, for they develop later into large cystic cavities, lined with epithelium."

2. Abscesses.

I. Acute

II. Chronic
- (*a*) Blind.
- (*b*) With a Sinus.
- (*c*) Discharging through a root canal.

I. Acute Abscesses.

I. An acute alveolar abscess may be regarded as a reaction to strong injurious agents and the injury produced by them. In the

periapical tissues, this is usually brought about by the invasion of a large number of pyogenic bacteria into these tissues from the root canal.

An initial temporary constriction of the arteries is followed by a dilatation of the vessels with a more rapid flow in the blood stream. Then the blood flow becomes slower, although the dilatation continues. The veins also show an increase in their calibre with increased flow of blood followed by a slowing in the rate of the flow. In some of the capillaries and veins, flow of blood may entirely cease.

Following a margination of leukocytes along the vessel-walls, into the spaces between the principal fibres of the pericementum which pass from the cementum to the alveolar wall, there takes place an emigration of these leukycytes, which by altering their contours and piercing the vessel-walls pass out into the lymph-spaces of the periapical tissues. The polymorphonuclear leukocytes play the most important part in this type of inflammatory reaction and are the first to pass through the walls of the vessels into the surrounding tissues. In the capillaries, red blood-cells as well as the leukocytes pass through between the endothelial cells, but their movement, unlike the leukocytes, is entirely a passive one.

Some of the constituents of the blood plasma pass from the blood-vessels, with the leukocytes and red blood-cells. The function of this serum is to dilute the toxic substances formed by the bacteria and to neutralize their action. At this stage, due to these changes, there is a great increase in the thickness of the pericementum and the tooth is somewhat elongated in its socket. This is known as *acute pericementitis*. If the conditions continue, destruction of tissue takes place and there is noted between the fibres, small collections of pus. If the inflammatory reaction is prolonged, disappearance of the principal fibres occurs by solution, due to the action of ferments secreted by the polymorphonuclear leukocytes. After the loss of the lamina dura of the alveolar process, the pus thus formed infiltrates the cancellous part of the bone, and an exit to the outer bony plate is provided by one or more Haversian canals.

When the pus reaches the periosteum, one of two events may happen. In some cases, this tissue is lifted from the bone, the pus collecting underneath, in others, by necrosis and tissue solution, a sinus is established through the adjacent tissues to the surface. If the pus be evacuated and the injurious agents eliminated, it remains for the cells of the parts to fill in the areas lost in the process. Fibroblasts and vascular endothelium are formed and the space is quickly filled up. The fibroblasts, as time passes, become more fusiform in shape and elaborate processes which anastomose. The collagen fibrils increase in amount, many of the fibroblasts and blood-vessels disappear, the connective tissue formed becomes denser until in the final stage, it is known as scar tissue (fig. 125).

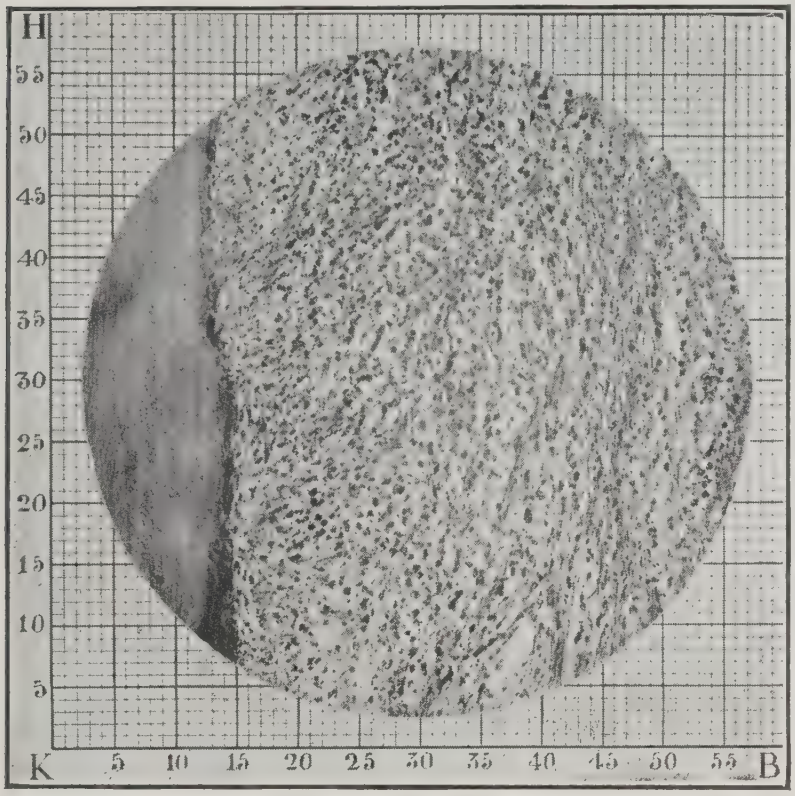


Fig 125— x 188. Acute alveolar abscess.

H K 30 x K B 14, Border of cementum.
H K 35 x K B 27, Polymorphonuclear leukocytic infiltration.

II. *Chronic.*

Chronic periapical abscesses which remain attached to the tips of the roots after extraction, when viewed macroscopically, present the appearance of small, soft, red or yellow swellings. Upon incision, they prove to be thin connective-tissue capsules or sacs containing pus. Those cases which present no route of discharge of pus are known clinically as blind abscesses, and frequently are formed from granulomata which have undergone purulent degeneration. Where a direct route of discharge is present, in its most common form there is a sinus extending from the abscess to the surface. Quite often, however, the pus finds an exit through a root canal which has offered a path of least resistance. The abscess capsule is made up of bundles of connective-tissue fibres, the arrangement of which is similar to that found in the granulomata. Projections of this connective-tissue capsule extend into the medullary spaces of alveolar bone (figs. 126 and 127).

3. Root-Cysts.

Development of the fifth class of granulomata, previously described, in which there are noted areas containing fragments of degenerated cells and surrounded by squamous epithelium, progresses by dilatation of these lumina. Such dilatation is believed to take place by an increase in the liquid content of these lacunae whereby

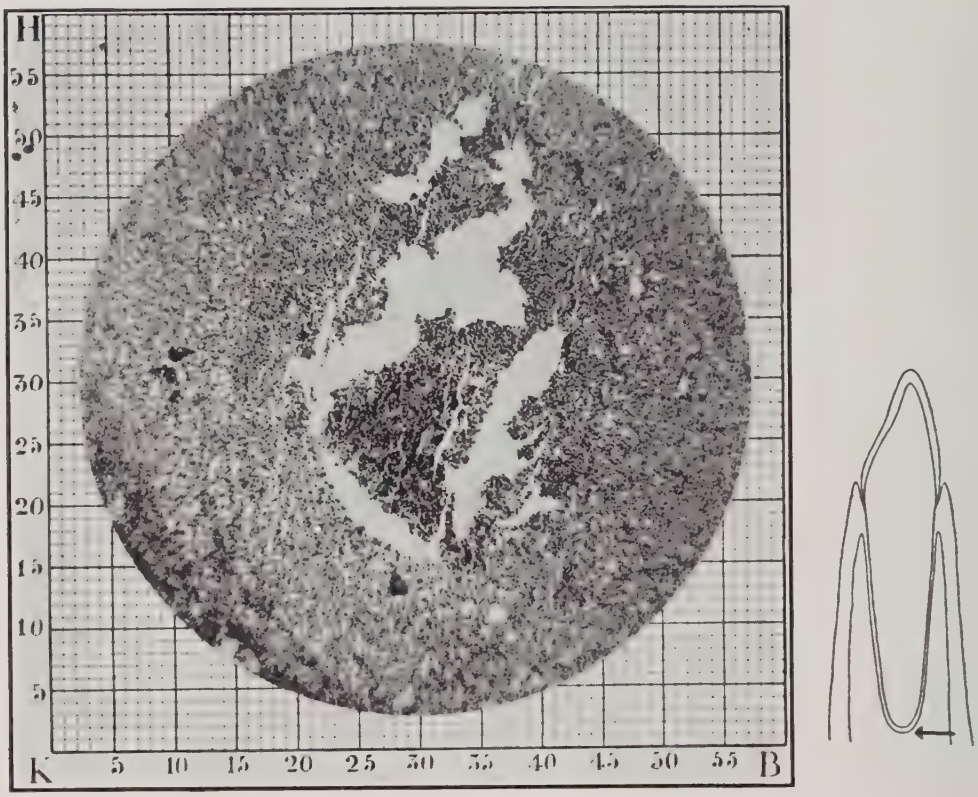


Fig. 126— x 94. Chronic alveolar abscess.

H K 35 x K B 35, Pus.
H K 40 x K B 11, Granulation tissue.

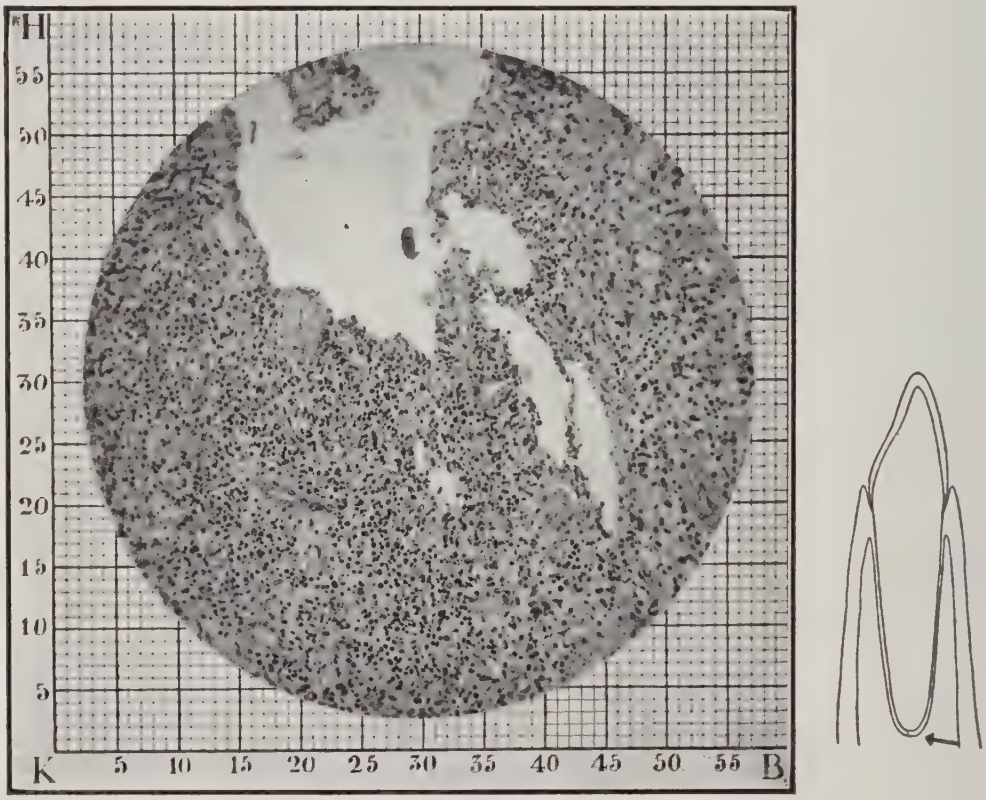


Fig. 127— x 94. Chronic alveolar abscess.

H K 45 x K B 25, Space originally occupied by pus.
H K 20 x K B 27, Granulation tissue.

the connective-tissue layer is pressed toward the periphery. Thus, the outer capsule of the cystic sac is formed. The epithelium is displaced and changed into the smooth membrane of the cystic sac (figs. 128, 129, 130). As the cyst becomes larger through increase of the fluid content, the surrounding bone becomes progressively thinner. Fischer believes that the cystic fluid is due to the mucinous softening of the surrounding connective tissue; that the space lined with epithelial cells is filled by a fluid thus formed which passes through the epithelial layer. It is also his belief that an inflammatory exudate may diffuse through the cellular wall.

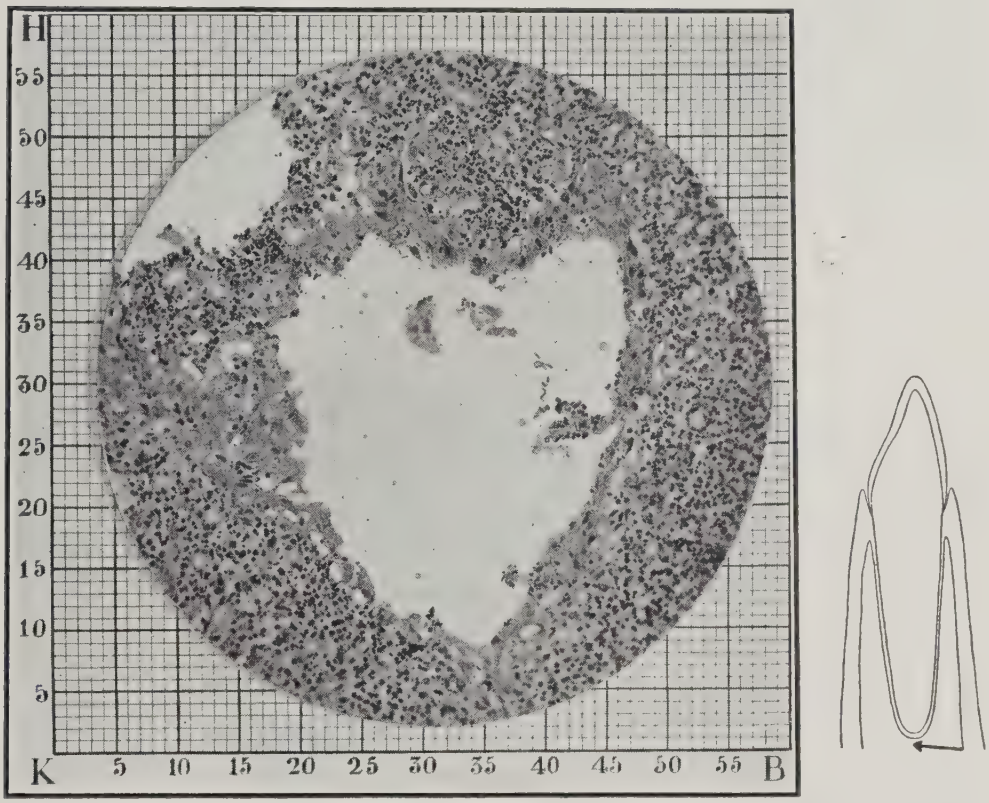


Fig. 128— x 188. Root-cyst.

H K 30 x K B 19, Epithelial lining of cyst.
H K 30 x K B 30, Cystic fluid.
H K 15 x K B 17, Granulation tissue.

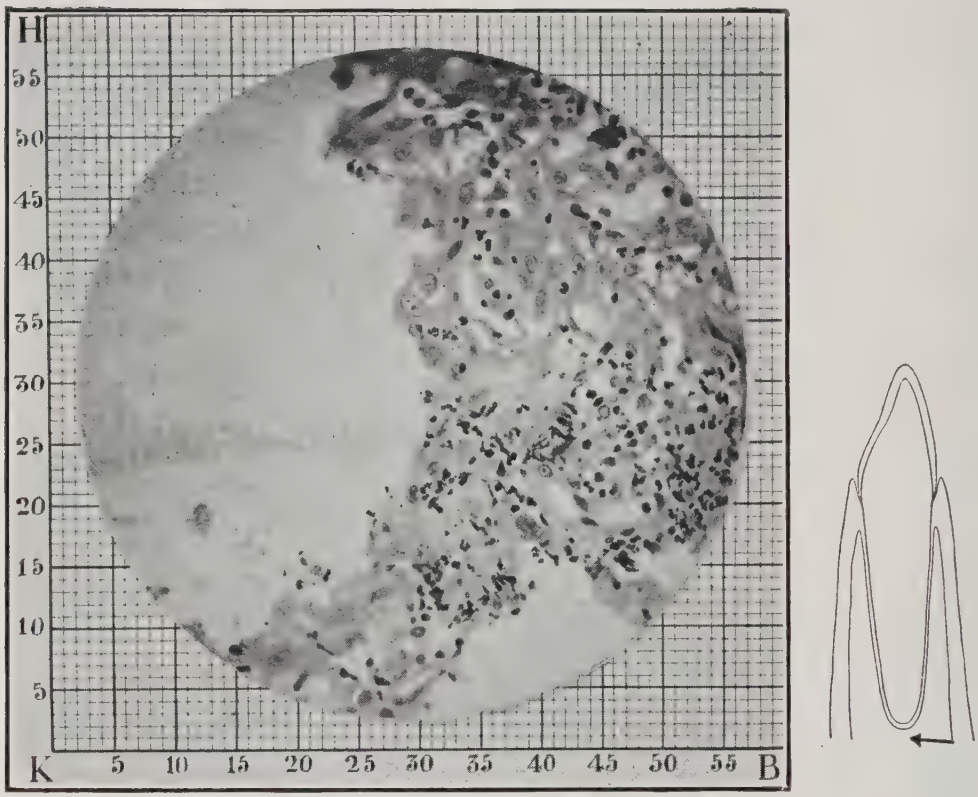
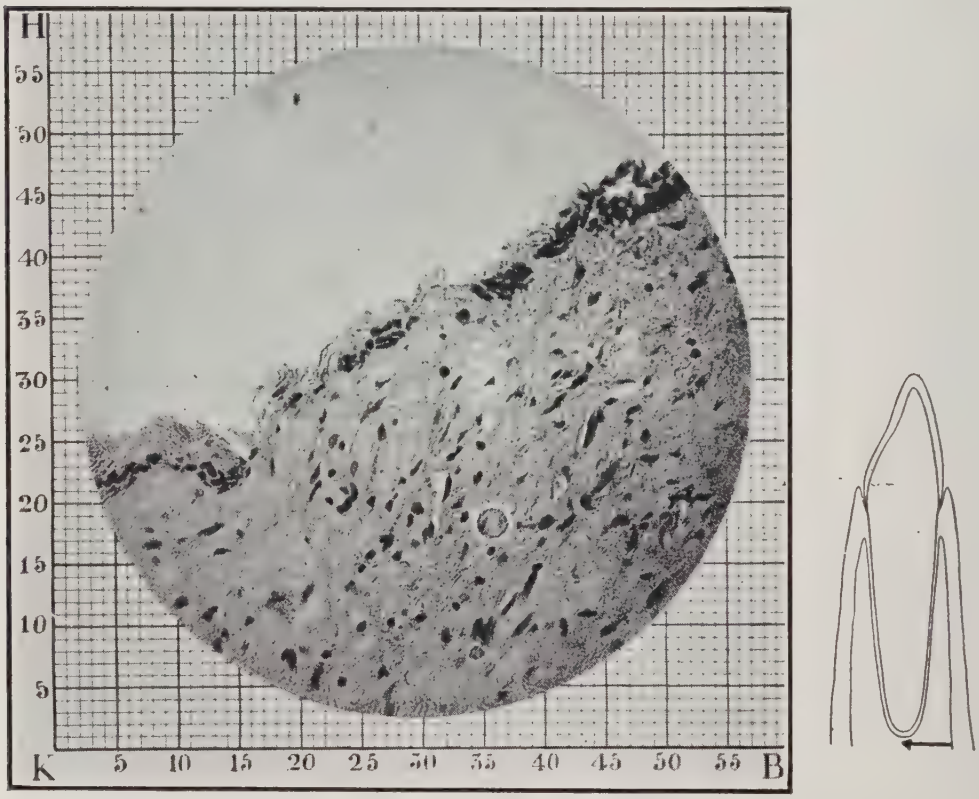


Fig. 129— x 500. Root-cyst. High magnification of the specimen shown in the preceding illustration.

H K 37 x K B 29 } Epithelial cells of the lining of the cyst.
H K 11 x K B 24 }
H K 30 x K B 20, Cystic fluid.



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HAROLD KEITH BOX.